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Archives of Dermatology and Syphilology

VOLUME 16 DECEMBER, 1927 NUMBER 6

THE AMERICAN ORIGIN OF SYPHILIS

WITH CITATIONS FROM EARLY SPANISH AUTHORS COLLECTED BY DR. MONTEJO Y ROBLEDO

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Scholars have expended prodigious amounts of labor and of learning in the endeavor to find descriptions of syphilis in ancient writings. Egyptian papyri, the Old Testament, early books in Sanskrit and Chinese, in Greek, Latin and Arabic have been searched for the desired evidence. Some students profess to have found such evidence. But their devoted efforts seem not to have convinced the rest of the world, for we find the subject still being debated. Nearly all students appear to be agreed, however, on one point. As the story has been told many times, we shall repeat it with the utmost brevity, and only in order to make clear that which is to follow.

In 1494 and 1495, Charles the Eighth of France conducted a campaign against Naples, leading an army southward through Italy. Naples fell to the French on Feb. 22, 1495. At Naples, unmistakable syphilis made its appearance almost in the form of a devastating epidemic. The French called it the Neapolitan Disease and the Italians called it the French Disease, as will appear in some of the contemporary writings quoted farther on. It is possible, as several have suggested, that other communicable diseases may also have prevailed among the armies or the population, and that the effects of such diseases may have been attributed to syphilis in part. The returning French army let loose on the rest of Europe a lot of wandering ruffians and dissolute characters who, in a short time, made syphilis a well known malady and a terrible problem. It was still generally called the French disease or morbus gallicus. For instance, that engaging rascal, Benvenuto Cellini, informs us that he had the French Evil. In Spain, the disease was, as we shall

see, usually called bubas. It acquired the name syphilis in the years succeeding 1530, when Fracastor produced his Latin poem in which the shepherd Syphilus is a principal character. This very multiplicity of names according to countries (for there were many other such names) that came into use about 1493 and in succeeding years, Montejo argues, is an indication that syphilis was a disease previously unknown to the people of the countries that named it after their neighbors.

The view that we have stated as being the usual one has, however, been energetically opposed by the profound German scholar, Sudhoff. The use of morbus gallicus (or mal franzoso, mal français, Franzosenkrankheit, grosse vérole, etc.) for skin diseases that probably were chiefly syphilitic had been current in southern Europe for some years prior to the discovery of America, according to Sudhoff. Furthermore, he maintains that there was no epidemic of syphilis at the time of the campaign against Naples, though other diseases doubtless were present in epidemic form. Sudhoff agrees that syphilis attracted much attention and was widely discussed from about 1494 and 1495.[1]

The sudden appearance of such a scourge which, if not hitherto totally unknown, was at least unknown to the vast majority of Europeans, could not fail to arouse inquiry as to its origin. Many fantastic explanations were offered, mostly involving some supernatural agency, as was the custom in those times. The possible importation of syphilis from America by the sailors of Columbus was also suggested shortly after the discovery. The coincidence in time between 1493 and 1495 is startling, but there is also almost as striking a coincidence with the dates of the voyages of the Portuguese along the west coast of Africa a few years prior to 1492. A controversy began then that has lasted down to the present day. In such a condition of affairs, we evidently need more facts and not more controversy. It is our purpose in this article to make some of the evidence obtained from early Spanish authors accessible to Americans who may not read Spanish.

At the International Congress of Americanists held in Madrid in 1881, a Spanish physician, Montejo y Robledo, presented a monograph in which he gave extracts from a number of Spanish historians and scientists, most of whom were contemporary with Columbus or nearly so.[2] Montejo's work, although of intense interest, was practically over-

1. Sudhoff, Karl: Essays in the History of Medicine, translated by Fielding H. Garrison and others, New York, Medical Life Press, 1926, pp. 259-274. Garrison, Fielding H.: An Introduction to the History of Medicine, Philadelphia, W. A. Saunders Company, 1924, pp. 128-130. Garrison summarizes Sudhoff's discoveries of original manuscripts and gives numerous references to the literature.

2. Montejo y Robledo: Fourth Session, International Congress of Americanists, Madrid, Actas, vol. 1, p. 331, 1881.

looked, until it was reviewed by Iwan Bloch, who also made it available in abstract form to English readers.[3]

American students are similarly indebted to Pusey, who gives a brief synopsis of the same material in his "Syphilis, A Modern Problem."[4] As far as we can learn, neither Montejo's monograph nor the works of the historians quoted by him have been translated into English. We are deeply indebted to Montejo for having unearthed from the early Spanish records, often comprising many volumes, the portions that bear on the history of syphilis. To one not living in Spain and not familiar with the Spanish language, the labor would have been prohibitive. Montejo's monograph is too long (about eighty pages) to publish in translation in its entirety; furthermore, the greater part of it is taken up with a discussion and with comments of his own. We propose giving in full (with one exception) translations of his citations, six in number, that purport to give the history of the origin of syphilis in America. One of his authorities, Oviedo, has long been known and often quoted. His last and best witness, Ruy Díaz de Isla, was, as far as we can learn, unknown until brought to light by Montejo. The translations have been made as nearly literal as possible. It must be remembered that we are dealing with Spanish of the early sixteenth century for the most part, and that it differs from modern Spanish, though not in the same degree, as English of that period differs from modern English. The punctuation, the spelling, the accentuation and the use of capitals are often faulty and add to the difficulty of translation. A few passages have been encountered in which the meaning is obscure and that might admit of a different rendering, but none of those involved any point of importance to our subject.

Montejo begins his monograph with a discussion of the origin of the Spanish word bubas, a discussion that is of some importance because the Spanish historians quoted by him usually designated syphilis by this name. The word buba, he states, was a noun, possibly of Arabic origin, already in use at the time of the discovery of America, and meaning a scab on a wound or a little tumor of matter. When syphilis appeared as a new disease with multiple visible lesions, the plural form bubas was given to these lesions; eventually bubas came to be employed in Spain in the singular sense as the name for syphilis.

Montejo then gives the results of his examinations of various dictionaries, dating from the sixteenth and seventeenth centuries, of Indian languages of South America, Yucatan and Mexico. He finds that in a

3. In Power, D'Arcy, and Murphy: A System of Syphilis, London, Oxford University Press, 1908, vol. 1, chapter 1.

4. Pusey, William Allen: Syphilis, a Modern Problem, Chicago The American Medical Association, 1918.

considerable number of these languages, the Indians had their own words for bubas and its derivatives.[5] From this fact, Montejo argues that syphilis must have been an ancient and widespread disease in those regions of America.

We find this part of his work the least convincing on account of the possible confusion of syphilis with yaws (frambesia) in some of these languages. According to Castellani and Chalmers,[6] at the present day bubas is often applied to yaws in South American countries. Strong, Shattuck and Wheeler[7] have recently reported that in Brazil bouba is the term applied to yaws, but that it is sometimes used for other forms of ulceration. Howard[8] states that buba is employed as a name for yaws among the Swahili of Africa.

Yaws is also known as pian in the French Antilles, and has many other local names in Africa and in the East Indies. In studying the history of these diseases, it is necessary to be guarded so that yaws and syphilis may not be confused with each other. It would, however, be foreign to our aim in this paper to enter on a discussion of any possible relationship between yaws and syphilis. Students of languages will no doubt prefer to consult Montejo's work in the original. For our purposes, it is sufficient to state our opinion that the fact that various Indian languages had words equivalent to the Spanish bubas or the French vérole does not necessarily prove that syphilis was known to the Indians. But there can be no doubt that the historians that we are about to quote, writing shortly after the time of Columbus, did designate syphilis by the Spanish word bubas. We believe that the internal evidence of their narratives will make that apparent.

Montejo found one reference to syphilis, only a few lines in length, in the "Life of Christopher Columbus" written by his son Ferdinand.[9] This book includes the manuscript of the hermit Roman Pane of the order of St. Jerome under the year 1495, when the second voyage of Columbus was made. The fragment quoted refers to fables of the

5. When the vocabularies were compiled by French priests, the words vérole (pox) and grosse vérole (large pox) were used in the same way as the Spanish bubas, smallpox being indicated by petite vérole, as it now is.

6. Castellani and Chalmers: Manual of Tropical Medicine, ed. 3, London, Balliere, Tindale and Cox, 1919, p. 1535.

7. Medical Report of the Hamilton Rice Expedition to the Amazon, Boston, Harvard University Press, 1926, p. 16.

8. Howard: J. Trop. Med. & Hyg. **18**:25 (Feb. 1) 1915.

9. Historia del Almirante de las Indias Don Christoval Colon, printed in Madrid in 1749, first column, page 63. This history underwent many vicissitudes. It was translated into Italian, and the original having disappeared, it was again translated from Italian into Spanish. The latter translation is the one studied by Montejo.

Indians, chiefly of the island of Haiti. Whether the name "French disease" was used in the original or was applied by one of the translators is not stated and probably is not known.

"They say that Guagagiona being in the land where he had gone, saw a woman whom he had left on the sea, from whom he had great pleasure, and immediately he sought many lotions to cleanse himself, on account of being plagued with the disease that we call French; and afterwards he betook himself into a Guanara, which signifies a place by itself, where he recovered from his ulcers."

In reading the quotations from early Spanish authors, it is necessary to keep in mind the fact that Christopher Columbus died having the conviction that he had encountered the coast of Asia or its outlying islands. The idea that an entire new continent had been discovered was one that entered men's minds slowly as evidence piled up in subsequent years. However, Oviedo, who is the next authority cited, must have had a clear notion of the position of America; for years, he was in the tropical parts of America, and wrote his history long after Magellan's crew had sailed around the world. Oviedo's accuracy as a historian has been doubted by some and upheld by others; he was certainly a gossip. Las Casas is said to have declared that his books had as many lies as there were pages. However, as Montejo points out, in respect to the origin of bubas, Las Casas, whom he also quotes, in general confirms the statements of Oviedo; in fact, the two accounts are singularly alike in phraseology, though differing in detail.

CITATIONS FROM SPANISH AUTHORS

Gonzalo Fernández Oviedo y Valdés [10] (1478-1557) in his "General and Natural History of the Indies," printed in its complete form after the middle of the nineteenth century by the Royal Academy of History, Madrid, wrote:

> These facts have been recalled in order to point out the time when Columbus arrived at the Court, as to which I speak as an eye-witness, because as a boy I was a page at the siege of Granada, and I saw the town of Santa Fe [11] founded in that army, and afterward saw the Catholic King and Queen enter the city of Granada when it was delivered up to them, and saw the expulsion of the Jews from Castile, and was in Barcelona when the King was wounded as I have said; and saw the admiral *Don Christobal Colom* come there with the first Indians that went there from these parts on the first voyage of discovery. So that I do not speak from hearsay in any of these four things but from experience; although I am writing them down from memory, or more exactly, by referring to my notes which were written at the time when the things described in them happened.

10. Oviedo: Historia general y natural de las Indias, Book 2, chapter vii, p. 28.

11. Santa Fe was the camp or city built by the sovereigns for their army at the siege of Granada.

Book II, Chapter III, page 50:

These Christians, first settlers of this island, suffered moreover much difficulty with the chigoes, and very cruel pains and torments from the disease of buas,[12] (for the Indies are the place of their origin), and I do well to say the Indies [Spanish Indias] both for the country where this affliction is so characteristic and for the Indian women of these parts, by communication with whom this plague was transmitted to some of the first Spaniards that came with the admiral to discover these regions, because as it is a contagious disease, that could very well happen. And from them, when they had returned to Spain and had disseminated this affliction in that country, from there it was transmitted to Italy and other places as I shall tell later, etc. And I shall not forget the efts, snakes and lizards that are in this country; and I shall speak of the torment from the chigoes, and of the detestable disease of the buas, with which will be given an account of the eleven things touched upon above.

Many times in Italy I did laugh, hearing the Italians say the French disease and the French calling it the disease of Naples; and in truth both would have hit on the right name if they had called it the disease from the Indies. And that this is the truth, can be understood by this chapter and from the extensive experience acquired now with the lignum vitae and with the guaiac wood with which especially the horrible disease of the buas is relieved and cured better than with other medicine, for the divine clemency is so great that wherever it allows us to suffer for our sins it grants that there should be found likewise the remedies for this suffering. [These two trees are discussed in Book V, Chapter ii, etc.]

In the preceding chapter I said that Columbus returned to Spain in the year one thousand four hundred and ninety-six[13] and such is the truth; after which event I saw and spoke with some of those that came back with him to Castile, as well as with the Commander Sir Pedro Margarite and to the Commanders Arroyo y Gallego and Gabriel de Leon and Juan de la Vega and Pedro Navarro, chamberlain of Prince Don Juan, my master, and to most of those who are included in the list of servants of the royal house who came on the second voyage of discovery of these regions. From those and others I heard many things of this island and of what they saw and suffered and learned from the second voyage, besides what information I gained from them and others of the first voyage, as well as from Vincente Yañez Pinçon who was one of the first pilots of those three Pinçon brothers of whom mention has been made; for I kept up my friendship with the latter until the year one thousand five hundred and fourteen when he died. I gained information also from the pilot Hernan Perez Matheos, who at the present time lives in this city, who took part in the first and third voyages that the first Admiral Don Cristóbal Colom made to these Indies. And also I have obtained news of many things of this island from two noblemen who came on the second voyage of the admiral that at the present day are here and live in this city, who are Juan de Rojas and Alonso de Valençia, and from many others who as eye witnesses of what has been said in regard to this island and its affairs gave me minute accounts. And more than anyone else of all those that I have spoken of, the Commander Sir Pedro Margarite, a leading man in the

12. Montejo says that buas is sometimes used in the same way as the form bubas.

13. Referring to the second voyage.

royal household, and the Catholic King had him in good esteem. And this knight it was that the King and Queen took for their principal witness and to whom they gave most credit for the things that had taken place on the second voyage to which reference has already been made. This knight, Sir Pedro, went about suffering so much and complaining so much that I also believe that he had the affliction that they are wont to have who are affected with this torment but I did not see any buas on him. And after a few months of the above named year, one thousand four hundred and ninety-six, this disease began to be felt among certain courtiers; but in the beginning this disease was among low persons and of little authority, and so it was believed that they acquired it by consorting with public women and from such lustful behavior, but later it extended among the people of higher rank and greater importance.

Great was the wonder produced in all that saw it, not only because the disease was contagious and horrible, but because many died of this disease. And as the disease was something new the physicians did not understand it and did not know how to cure it, nor were there others with experience to advise about such a disease. The next thing was that the great captain Gonçalo Fernandez de Córdoba was sent to Italy with a large and goodly army by command of the Catholic Sovereigns and as their captain general in support of King Ferdinand, second of that name in Naples,[14] against King Charles of France whom they call the fat-head; and among those Spaniards some were touched with this disease, and through the women of bad conduct and life it was communicated to the Italians and French. Then, as such a disease had never before been seen there by any of them, the French began to call it the disease of Naples, believing it was peculiar to that kingdom, and the Neapolitans, believing that the French had brought that affliction, called it the French disease,[15] and it has been called so ever since in all Italy; because until King Charles passed through there such a plague had not been seen in those lands. But the truth is that this disease was transmitted from this island of Haiti or *Española* to Europe as stated; and it is very common among the Indians here and they know how to cure themselves and they have very excellent herbs and trees and plants appropriate to this and other diseases such as the guaiac (which some people prefer to call hebeno) and the lignum vitae, as will be mentioned when the trees are treated of. And so of the two dangerous plagues that the Christians and new settlers of these Indies suffered from, and today some are suffering from, that are native diseases of this land, this of the buas is one, and the one that was transferred and transported to Spain and from there to the other parts of the world, without ceasing to exist here. So continuing the subject of the diseases of the Indies, I will tell of the other disease that manifested itself, the chigoes.

There is in this island and in all these Indies, islands or Terra Firma, the disease that I have spoken of, buas, and the other that they call the chigoes.

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14. The king of Naples as well as the king of Spain was named Ferdinand (the second).

15. At this point Oviedo differs from Las Casas and Ruy Díaz de Isla. The army of Cordova was sent in 1496. Other historians state that syphilis appeared at Naples when the French army marched southward early in 1495, and it was very soon called the French disease. Ruy Díaz de Isla, who is the last witness produced by Montejo, declares, as will be seen, that Spaniards from Barcelona having bubas were in the French army.

Las Casas[16] (1474-1566), who for many years was in tropical America, wrote:

Two things there were and are in this island that were very grievous for the Spaniards in the beginning: one is the disease of the bubas that in Italy is called the French malady; this, let it be known in truth, was taken from this island, either when the first Indians left at the time when the Admiral D. Cristóbal Colom returned with the news of the discovery of these Indies, which men[17] I myself saw soon afterwards in Seville, and these were in a position to communicate it to Spain, by infecting the air or in other ways [what a pointed warning!]; or when some Spaniards having already contracted the disease went on the first return voyage to Castile, and this could have happened between the years 1494 to 96; and because at this time King Charles of France, whom they call the Bighead, passed with a great army into Italy, to take Naples, and that contagious malady was in that army—for this reason the Italians thought they had caught it from them, and from then on they called it the French disease. I myself sometimes endeavored to inquire of the Indians of this island if this malady were very ancient on it, and they answered yes, before the Christians came to it, without having memory of its origin, and of this none ought to doubt; and good appears in it also, since divine Providence provided for it its proper medicine, that is, as above we stated in chapter 14, the guaiac tree. It is a thing well verified that all the incontinent Spaniards that did not have the virtue of chastity on this island were contaminated by it [recall, gentlemen, the clear warning that I brought to your attention a few moments ago], and out of a hundred perhaps not one escaped except when the other party never had had it.

The Indians, men or women, that had it were little affected by it, almost as little as if they only had smallpox; but for the Spaniard the pains from it were intense and continuous torment, particularly up to the time that the bubas appeared. The other thing that afflicted some Spaniards in the beginning, was what the Indians called niguas;[18] these are a certain species of flea, and they jump like fleas, and they are so small that they can scarcely be seen.

Sahagún[19] was born in Spain about 1499 and went to Mexico in 1529, where he died in 1590. Mexico was then called New Spain. In his "History of the Affairs of New Spain," he says:

Paragraph V. Of other diseases and the antagonistic remedies.—The disease of the bubas is cured by drinking the water of the herb named tletlemaitl and taking baths and putting on the bubas the powder of the herb called tlacuecuetzal, or the filings of copper. These bubas are of two kinds:[20] one is very filthy which is called tlacaçol nanavatl, and the other of less severity, which is

16. Bartolomé de las Casas or Casaus, Bishop of Chiapa: Historia de las Indias, vol. 5, p. 233, published in 1875-1876 by the Marqués de la Fuensanta del Valle and Don José Sancho Rayón.

17. I. e., the Indians. Columbus brought Indians back with him to Spain on the first as well as on other voyages.

18. Chigoes.

19. Bernardino de Sahagún: Historia universal de las Cosas de Nueva España, chapter 18, book 10.

20. It is possible that one of these forms was yaws and the other syphilis. Castellani and Chalmers do not mention yaws as occurring in Mexico today and further state that it is rarely acquired at altitudes above 800 feet.

called tecpilnanavatl and by another name, pochunanavatl, and these give great pains, and cripple the hands and the feet, and are embedded in the bones; and when they come out the atolli [21] should be drunk, mixed with certain seeds named michivauchtli, or one will drink the water of the root called quauhtlepatli four or five times each day, and will take some baths, and if the sick person becomes crippled he will drink the water of the root named tlatlatlapanaltic and at last be bled. The remedies named are used for the other kind of bubas already named.

Montejo remarks: "With a purpose very different from that which impelled Bernardino de Sahagún to describe in detail in chapter 28 of his book 10 the diseases special and peculiar to the natives of Mexico, he confirms in chapter 2, book 7 of his history, the traditional, and without doubt the ancient, existence of the bubas in the great empire of the Aztecs, because in treating of the moon in the extremely curious cosmogony of the Aztecs, he says:

When the moon newly born appears like a bow of thin wire, it does not yet shine, but little by little it increases. In fifteen days it is full. And when at last it is full and climbs to the east at sunset, it looks like a mill-wheel, large, very round and bright red. And when as it rises it stands white and glittering, there appears what looks like a rabbit in its center, and if there are no clouds it shines almost like the sun in the daytime. After it is completely full it wanes little by little and declines until it becomes as it was when it began. They say then that the moon is already dying, that already it is fast asleep. When it arises with the dawn at the time of the conjunction they say the moon is already dead. The fable of the rabbit which is in the moon is this. They say that the gods made fun of the moon and threw a rabbit at its face and the rabbit remained marked on its face and thereby they darkened its face as with a weal. After which it arose to light the world. It was said that before there was day in the world the gods got together in the place that is called Teutioacan [that is, the pueblo of Sant Juan between Chiconauhtlan and Otumba], and the gods discussed, the ones and the others, who should undertake the task of lighting the world. Immediately to these speeches replied a god who was called Tecuciztecatl and said I will undertake the task of lighting the world. Later, the gods again spoke and said: Who will be the other? Immediately the gods looked at each other and conferred as to who would be the other, and no one dared offer himself for this office. All were afraid and excused themselves. One of the gods of whom nobody took any account and who was buboso (having buboes), did not speak but was listening to what the other gods were saying, and the others spoke to him and said to him: Be thou the one that lights it, bubosito (little one with buboes); and he out of good will obeyed their commands and replied: Willingly I undertake what you have commanded; so let it be, etc., etc.
farther on the expression "the scabs of the buboes" (las postillas de las bubas) is used.

We have omitted the rest of this citation, which is long and seems not important.

Francisco Hernández (1514-1578), physician to Phillip II, was sent to Mexico by Phillip, and remained there from 1570 to 1576, to study

21. Atole, gruel made by boiling Indian corn, is presumably the same word.

the natural history of that country. In his four books: "On the nature and virtues of the plants and animals that are recognized for use in medicine in New Spain, and the method and the modification and preparation required for their administration, with what Doctor Francisco Hernández [22] wrote in the Latin language, etc.," the following appears in folio iii:

Chapter XXXIII. On the Nanahuapatli.—Nanahuapatli, which means medicine for the bubas or French malady, which others call Palancapatli, because it cures ulcers, is an herb that has leaves with a certain roughness, and of evil appearance, long and like those of the common pinocela, the stem slim, short and rounded, and at the top of the stem it bears a flower like the camomile. The seed is sharp and biting, the root long and slim and abundant with filaments, it grows in temperate regions, such as the lands of Tepuztlan, it is warm and dry in a moderate degree and of a bitter, aromatic taste. Made into powder and dusted on the putrid ulcers, it heals them wonderfully, for which reason as has been said it was called by this name. It cures those who suffer from melancholia, and those who are bitten by the serpent called homorroes, and the people of Panuco call it mahuaquitliquin. Besides this, comminuted in water, or in some liquor that may be suitable, and being given as a beverage when and as may be suitable, it cures completely the disease that they call French or Neapolitan malady, consuming and excreting all the humors, ulcers and swellings that one usually finds on the body of those that suffer from this malady. From which is more apparent than the light of noon itself that in the wake of many sufferers from this disease of bubas; it came out of these West Indies, and from here was extended and communicated through different provinces and kingdoms of the world, since among these people this disease has its special, native and ancient name, which the other diseases do not have or very few of them.

The first printed edition of a work of Ruy Díaz de Isla [23] was a "Treatise on the serpentine malady, which in Spain is commonly called bubas, which was drawn up in the hospital of all saints in Lisbon." At the end it says that the treatise "was printed in the very noble and very loyal city of Seville, in the house of Domenico de Robertis, printer of books. Finished the twenty-seventh of September, MDXXXIX."

De Isla says:

It has pleased divine justice to give and send down upon us unknown afflictions, never seen nor recognized nor found in medical books, such as this ser-

22. Hernández, Francisco: Quatro libros: De la naturaleza y virtudes de las plantas y animales que están receuidos en el vso de Medicina en la Nueva España, y la Methodo, y correccion, y preparacion que para administrallas se requiere, con lo que el Doctor Francisco Hernández escribió en lengua latina, etc.: translated into Castilian by Fray Francisco Jiménez and printed in Mexico in 1615.

23. Tractado contra el mal serpentino: que vulgarmente en España es llamado bubas que fue ordenado enel ospital de todos los santos de Lisbona; fecho por ruy diaz de ysla. Fue impresso en la muy noble y muy leal ciudad de Sevilla, en casa de Dominico de Robertis impressor de libros.—Acabose a veinte y siete de Setiembre año de MDXXXIX.

pentine disease. And this appeared and was seen in Spain in the year of our Lord one thousand four hundred and ninety three in the city of Barcelona: which city was infected and consequently all Europe and the universe in all known and communicable parts: which disease had its origin and birth once and for all on the island that today is named Española: as has been found by very long and well proved experience. And this island was discovered and found by the admiral don Xrisptoual Colon, at present holding intercourse and communication with its people. And as it (the disease) is by its very nature contagious it easily attacked them and presently it was seen in the armada itself. And as it was a disease never seen nor known by the Spaniards although they felt pains and other effects of the aforesaid malady, they attributed it to the hardships of the sea or to other causes according to the fancy of each of them. And at the time that the admiral don Xrisptoual Colon arrived in Spain the catholic sovereigns were in the city of Barcelona. And when they went to give them an account of their voyage and of what they had discovered, immediately the city began to be infected and the aforesaid disease spread, as was seen later on through long experience. And as the affliction was not known and was so frightful, those who saw it hastened to do much fasting and prayers and alms in order that our Lord would be pleased to protect them from having such a disease. And afterwards in the following year of one thousand four hundred and ninety four the most Christian King Charles of France who then was reigning having gathered a great army passed into Italy. And at the time that he entered the country with his host many Spaniards infected with this disease[24] were in it and at once the camp began to be infected with the aforesaid malady, and the French as they did not know what it was thought it came from the atmosphere of the region. The French called it the disease of Naples. And the Italians and Neapolitans, as they had never had acquaintance with a like disease, called it the French disease. And from that time on as it continued to spread they gave it a name, each one according to his opinion as to how the disease had its origin. In Castile they called it bubas, and in Portpugal they called it the disease of Castile; and in the Indies of Portugal [i. e., East Indies] the Indians called it the disease of the Portuguese. The Indians of the Island of Española formerly just as we called pains and suppurations and ulcers bubas they called the disease Guaynaras; and hipas and taybas and icas. I call it the serpentine disease of the island of Española, in order not to depart from the custom according to which everybody gave it the name which it appeared to each one that the disease brought from its source; and consequently they called it, the French disease of Naples and the Italians the French disease and the Portuguese the disease of Castile; and the Indians of Arabia, Persia and India (East Indians) the disease of Portugal, as has been said: and as to naming this malady the serpentine disease it is because according to its loathsomeness I do not know anything to which I could more naturally compare it than to the serpent: for as the serpent is an ugly, fearful and horrid animal so this disease is ugly, fearful and horrid: a grave disease that suppurates and corrupts the flesh: and breaks and rots the bones and disrupts and contracts the nerves[25] and consequently I give it that name.

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24. This piece of evidence, that Spaniards who acquired syphilis in Barcelona were in the army of Charles VIII that went to Naples, doubtless as mercenary soldiers, Montejo regarded, in our belief with justice, as one of the choisest prizes of his researches.

25. It would be interesting to know what ideas of anatomy and physiology de Isla had; his notions were, of course, rudimentary according to our standards. He was writing about 100 years before Harvey discovered the circulation of the blood.

And knowing that this malady had its origin in olden times on the island of Española, and as it sprang from there, I give it the name, Serpentine disease of the island of Española, because from there the whole world was infected. Nevertheless any one may call and name this disease by the name that he likes as all the nations of the universe have done: for as Galen said I do not care about names: let the curative designs be right and good.

First edition, chapter 13, first face of folio 63.

In the first chapter it is told how this malady came from the island of Española, and many doubt it and hold that it appeared for the first time in the army of King Charles of France in the year 1494 and on this I have said enough in the same chapter, but I wish to give a reason so that among the discerning it may be seen clearly, and so I say that in the year 1504 there were given me in writing all the remedies that the Indians used for this disease as I have them written, as well with the guaiac as with the mapuan and with the tuna: now if that rude people was right about the treatment by which the disease is properly remedied and healed it follows that the disease was prevalent among them for a long time so that they had regulated both the drinking of water and the diet and the time when they should keep from women, as well as protection from dampness and the air, while in truth since this sickness has prevailed among ourselves no one of these things have I seen controlled down to today, neither mercury nor wine nor our habits; never to date have I seen any authoritative writing wherein may clearly be found the cure for this disease whether among Christians or among Moors and Gentiles of all the communicable parts: and as these people although they were the most stupid ever seen, were completely familiar with its cure and regulation, it is evident that because the disease always prevailed among them, for that reason, they knew the cure, being persons who had minute knowledge of the disease. For if this were not so, many other peoples much wiser than they would have found the cure for this disease for which reason all erroneous argument that may be made concerning the aforesaid may be given up. For I have had long experience and have cured persons who had it in the aforesaid armada and cured persons that were afflicted in Barcelona and I could quote many testimonials that are omitted: because the above seems to me to suffice abundantly for the case and those who wish may read more in the chapter on the cure by the *palo* and there may find more particulars.[26]

A second printed edition [27] of the same author, was entitled "Treatise called Fruit of All the Saints: against the Serpentine Malady Originating in the Island of Española, made and ordered in the great and famous Hospital of All the Saints of the noted and very celebrated City of

26. It is singular that the argument made by Díaz de Isla in the preceding paragraph, partly fallacious on account of the worthlessness of guaiac as a remedy for syphilis, should have been used also by Oviedo and Las Casas. Possibly the letter writers may have read the book of Ruy Díaz de Isla.

27. Tratado llamado fruto de todos los Santos: contra el mal Serpentino, venido de la ysla Española, hecho y ordenado en el grande y famoso hospital de Todos los Santos de la insigne y muy nombrada ciudad de Lisboa. Por el muy famoso maestro Ruy diaz de ysla. Vecino de la nombrada y gran ciudad de Sevilla.

Lisbon. The printing of this treatise was completed in Seville, Nov. 28, 1542, that is to say, three years and two months later than the first edition (Montejo).

On folio 76 of the edition of 1542 (second printed edition), he says:

——because of all this, I have long experience in curing persons who had it in the aforesaid armada, the first, when they discovered this land, in which came many people having them [i. e., bubas] and I did cure persons that were suffering in Barcelona before King Charles of France passed into Naples, and we could quote many testimonials that are omitted.[28]

The original manuscript of Ruy Díaz de Isla, which is preserved in the National Library of Madrid, is guarded like "leaves of gold." As it was dedicated to King Manuel of Portugal, who died in 1521, it must have been written before that date. The title is "Treatise called Fruit of All the Saints against the Malady of the Island Española done by Master Roderigo de Isla, Surgeon, Citizen of Lisbon, for the common and general profit of patients sick with the same disease that is vulgarly called Bubas."[29]

The original manuscript contains a paragraph of exceptional importance that is not found in the printed editions. The omission may have been made by the advice of the protomedicos (court physicians) who may have read the manuscript or for some other reason that cannot now be divined. (No folio number for this citation is given.)

As has been found by very long and well proved experience and as this island was discovered and found by the Admiral Dom Cristoual Colon at present holding intercourse and communication with the Indies. As it is of its very nature contagious, they got it easily: and presently it was seen in the armada itself in a pilot of Palos who was called Pinçon and others whom the aforesaid malady kept attacking. And as it was a secret disease never seen ——, etc.

Except for the statement concerning Pinçón, the citation is nearly identical with the early part of the first quotation from Ruy Díaz de Isla. Of the three Pinçón brothers who were with Columbus on his first voyage, probably Martín Alonso Pinçón, commander of the Pinta, is referred to in the foregoing paragraph.

The Pinta separated from the Niña on the return voyage and landed at Bayonne, a small town at the mouth of the Vigo river in Spain (not

28. This citation from the second printed edition is given to call attention to the title which explicitly states that the malady originated in the island of Española or Haiti; and to the assertion of Díaz de Isla that he personally handled cases in Barcelona prior to the campaign against Naples.

29. Tratado llamado Fruto de todos los santos contra el mal de la ysla Española hecho por maestre Roderigo de Isla cirujano vezino de lisboa para comun e general provecho de los pacientes Enfermos de la semejante Enfermedad que vulgarmente es llamada Bubas.

at Bayonne in France). From there Martín Pinçón sent a message to the sovereigns, in the expectation of anticipating Columbus. When this Pinçón reached Palos, Columbus had already arrived there, and had been received with great rejoicing. Martín Pinçón was snubbed by the sovereigns, and died shortly after. His death was attributed to chagrin, but one infers from Ruy Díaz de Isla that the real cause may have been syphilis acquired in the island of Haiti.

One other point on which Montejo lays some stress in his discussion may be referred to briefly. Recognizing the possibility that *bubas* might have been introduced at some of the other ports touched at by the ships of Columbus, as well as at Barcelona, syphilis or *bubas* should perhaps have been recorded at some of them. Montejo states that at the Azores, where Columbus first stopped, and at Lisbon little communication of the sailors with the populace was possible. At Bayonne on the Vigo, where Martín Pinçón anchored, and at Palos where both Columbus and Pinçón landed, the towns were so small that prostitution must have been absent or present to a limited degree, so that infection of the population by a venereal disease present among the sailors was unlikely to occur. Montejo believes that sailors going from Palos to Seville probably did introduce bubas into Seville, and thinks that the first part of the quotation from Las Casas given in the foregoing probably has exactly that meaning.

Montejo's second citation from the first edition of Diaz de Isla (chapter 13, folio 63) seems to establish the fact that this Spanish physician was collecting notes on bubas in 1504, or only about eleven years after the alleged first appearance of syphilis in Europe. It seems to us that the citations do not require further comment and that it is best to let the evidence contained in them speak for itself.

THE USE OF TETRAIODOMETHENAMINE IN FLEXIBLE COLLODION IN THE TREATMENT OF DERMATOPHYTOSIS *

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We possess a convincing array of evidence that the squamous and vesicular dermatoses of the palms and soles, as well as of the webbed spaces between the toes and fingers, are due to a mycotic infection when they are not caused by an obvious external irritation. We have not been altogether fortunate in arriving at a thoroughly satisfactory therapeutic procedure for these cases. Iodine, sulphur, chrysarobin, salicylic acid, benzoic acid and many other drugs,[1] and, finally, the roentgen ray, severally and in combination, have been recommended as ameliorative of this condition.

When a procedure for eradicating the infection is selected, the depth in the epidermis at which the fungi are harbored must be considered. Manifestly, the deep epidermal vesicles in the somewhat calloused soles demand a more strenuous keratolytic agent to uncap and evacuate them than would the squamous type of lesion on the palms. And so, a specific procedure might advantageously be described in its application to the type of dermatitis evidenced.

The medicament and the manner of its use that we present are still other means of combating dermatophytosis. They possess theoretical values that have in great measure been borne out by clinical experience, particularly when applied to lesions of the squamous and superficial vesicular types. If it is recalled that fungi thrive in the presence of an adequate supply of oxygen and water, that moisture aggravates mycotic lesions of the skin, that the supply of oxygen to the surface of the skin can be suppressed only by application to that surface of an impermeable membrane, the possible advantages of the use of flexible collodion on lesions of such nature may be appreciated. Flexible collodion, U. S. P., which contains castor oil and camphor, forms a membrane impermeable to water, and the alcohol and ether tend to dehydrate the skin over which the collodion is applied. For lesions on the hands, exposed as they unavoidably are to constant irritation from

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* From the Harriman Research Laboratory, the Roosevelt Hospital.

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1. Schamberg, J. F., and Kolmer, J. A.: Studies in the Chemotherapy of Fungus Infections, Arch. Dermat. & Syph. **6**:746 (Dec.) 1922.

water, this form of dressing should be particularly advantageous. While we did not make clinical tests of the value of simple flexible collodion dressings, a procedure often used and reported on in dermatologic work, we do not wish to minimize the value of this feature of the procedure. Iodine as a fungicide should be most efficacious in the treatment of patients who have mycotic dermatoses, not alone for its high fungicidal coefficient,[1] but also because iodine, extremely volatile at ordinary temperature, may be fed to the skin as a gas and as such has a fair measure of penetrability. Iodine is bound by proteins and salts and rendered inert. Its caustic action on skin demands limitation of its concentration in a medicament. A constant stream of iodine acting over a long period of time, reaching the skin in fungicidal strength yet not irritating the skin is a desideratum.

We have attempted to supply a chemical which, until completely exhausted, should be capable of slowly and continuously giving off free iodine; this chemical is tetraiodomethenamine $(CH_2)_6N_4I_4$) (tetraiodo-hexamethylenamine). This compound, first described by Horton [2] in 1888, is an unstable iodine compound giving off free iodine vapor much after the manner of iodine crystals, only more slowly. The characteristics of its solubility are those of iodine. Thus it is soluble in alcohol and ether-alcohol. This compound, then, to the strength of 0.5 per cent in flexible collodion, has been used in a manner which we shall indicate in our discussion of the treatment of patients with mycotic dermatoses.

We believe that in offering still another chemical for use in therapy that the mere record of clinical experience is not enough for demonstrating the value of a drug, especially when thoroughly controlled tests in the laboratory can throw light on its value. We submit, therefore, the following studies particularly directed toward demonstrating the superiority of the action of tetraiodomethenamine over simple iodine for the objective sought.

It has been definitely established that iodine leaves a collodion membrane, and its use in collodion has been practiced for many years. Free iodine with equal ease though less rapidly, leaves a collodion membrane containing tetraiodomethenamine. To demonstrate this, a collodion membrane may be made which contains this compound; on allowing it to remain exposed, the yellow-brown color imparted to the membrane by the compound gradually fades and in time disappears, leaving the membrane colorless. A more satisfactory demonstration may be carried out as follows: Make a flexible collodion bag containing the compound; fill the bag with a solution of potassium iodide and add

2. Horton, H. E. L.: Ueber emige hexamethylenamine Derivate, Ber. d. deutsch. chem. Gesellsch. **2**:1999, 1888.

a few drops of starch water. The water within the bag soon becomes blue, indicating the passage of iodine into it.

The experiment outlined in table 1 was performed as a demonstration of the inhibitory (fungistatic) action on fungus growth of tetraiodomethenamine as compared to iodine. Varying concentrations of tetraiodomethenamine were added by constant volume (from 1 cc. to 10 cc. of mediums) to Sabouraud's mediums, and the mediums so contaminated were inoculated with *Microsporon lanosum*. The mediums so contaminated were slanted and hardened before being seeded and were incubated at room temperature. To render both the free iodine and the tetraiodomethenamine soluble in water, potassium iodide was added to each of the solutions, making necessary separate tests with solutions of potassium iodide to measure its particular influence. Since

TABLE 1.—*Influence on Growth of Microsporon Lanosum (at Room Temperature) on Sabouraud's Proof Agar in the Presence of Contaminating Solutions*

Contaminating Solutions		Dilutions as Parts Iodine per 100,000 of Solutions *						Control Cultures
		40	20	4	0.4	0.04	0.004	
(1) Potassium iodide	a	+	+	+	..	..	..	Dist. water +
	b	+	+	+	..	..	..	Fungus = + o
(2) Methenamine	a	c	c	c	+	+	+	1 cc. solution 1 = o
	b	c	+	+	+	+	+	
(3) Potassium iodide plus methenamine	a	o	o	+	..	..	..	1 cc. solution 2 = c
	b	o	o	+	..	..	..	
(4) Potassium iodide plus iodine	a	+	+	+	+	+	+	1 cc. solution 3 = o
	b	+	+	+	+	+	+	1 cc. solution 4 = o
(5) Potassium iodide plus tetraiodomethenamine	a	o	o	+	+	+	+	1 cc. solution 5 = o
	b	o	o	+	+	+	+	

* In tables 1 and 2, all five solutions in original concentrations were made containing, for the particular compound or mixture, their equivalent in solution (5) which was 0.5 per cent tetraiodomethenamine plus 5 per cent potassium iodide; therefore all dilutions are expressed in terms of the iodine content in solution five. + indicates growth; o, indicates no growth; c, indicates contamination. Growths were inoculated on April 28, 1926, and examined on May 12, 1926.

tetraiodomethenamine is an additive compound of methenamine and iodine, it was similarly necessary to ascertain the effect of methenamine if there was any; hence, the five solutions outlined in the table. A footnote in the table explains the manner of expressing the dilutions, all based on the use of equivalent concentrations and all dilutions referred to as of the iodine concentration in solution 5 in table 1.

Though iodine in solution 4 fails to inhibit growth in both a 40:100,000 and a 20:100,000 dilution, in those dilutions tetraiodomethenamine does have fungistatic action. We have in this an indication of a difference in manner of action between free iodine and the iodine in tetraiodomethenamine. The nature of this difference is shown in table 3. It may be noted, too, that a solution of potassium iodide plus methenamine is apparently as effective an inhibitor as solution 5. This is worthy of future study. Since in solution 5 tetraiodomethenamine

is chemically an additive product of methenamine and iodine, it may appear that the superiority of solution 5 over solution 4 is due to solution 3, and that the difference in the fungistatic strengths of these solutions is not a result of a difference in operation of the iodine element in those solutions. The data in table 2 eliminate this possibility. A study of the fungicidal actions of these same five solutions is outlined in this table. Bits of masses of fungi (the size of a pea) were soaked for forty-eight hours in 1 cc. of each of these solutions; they were then washed for forty-eight hours in sterile distilled water, seeded on mediums and grown at room temperature. Solutions 4 and 5 show the same fungicidal strength, though solution 3 gives evidence of a much weaker fungicidal action. A comparison of tables 1 and 2 discloses the fact that solution 4, simple iodine, while fungicidal in a dilution of 40: 100,000, is not fungistatic in that dilution. This seeming

TABLE 2.—*The Fungicidal Action of Several Solutions on Microsporon Lanosum*

1 Cc. Contaminating Solution	Period of Action Hours†		Dilutions as Parts Iodine per 100,000 of Solution *					
			400	200	40	4	0.4	0.04
(1) Potassium iodide	48	a	+	+	+	..	..	..
		b	+	+	+	..	..	..
(2) Methenamine	48	a	+	+	+	..	..	..
		b	+	+	+	..	..	..
(3) Potassium iodide plus methenamine	48	a	o	o	+	..	..	..
		b	o	+	+	..	..	..
(4) Potassium iodide plus iodine	48	a	o	o	o	+	+	+
		b	o	o	o	+	+	+
(5) Potassium iodide plus tetraiodomethenamine	48	a	o	o	o	+	+	+

* Before masses of fungus were transferred to mediums they were washed in 10 cc. distilled water for forty-eight hours.

† Control Culture: Fungus in distilled sterile water for forty-eight hours gave growth on b ooo +++ culture.

paradox underlies the whole difference between the actions of simple iodine and tetraiodomethenamine, and demonstrates the superiority of the latter as a fungicidal medicament.

In experiment 1 (table 1), the tested solutions were added to a protein mixture (medium). In experiment 2 (table 2), the fungi were soaked in the pure test solutions. In the former case, the iodine of solution 4 was at once taken up in great measure by the protein of the mediums and rendered inert; this did not occur in the iodine in solution 5; in table 3 a clearly defined experiment is outlined which shows this difference in action between the two solutions of iodine. The iodine from tetraiodomethenamine is slowly given up, and its disappearance from a protein salt mixture such as blood serum proceeds at a slower rate than does iodine in simple form. In dermatophytosis one is dealing with a growth of fungus in a medium with a high content of protein and salt, the preference for the organic form of iodine over

simple iodine in the treatment of these dermatoses is, we believe, established by the data in table 3.

At the onset we treated the lesions by applying 0.5 per cent tetraiodomethenamine in flexible collodion twice daily, painting it on with cotton swabs or a brush for from one week to ten days. At the end of this time, the membrane was removed with ether, the lesions inspected and the applications repeated as necessary in the same fashion until the

TABLE 3.—*Rates of Disappearance (and Influence of Dilution) of Free Iodine from Iodine and Tetraiodomethenamine Solutions in the Presence of Diluted Blood Serum*

Iodine Cc.	Blood Serum 10 Per Cent Cc.	Water Cc.	Contact Time	Titration 0.005 N. $Na_2S_2O_3$ Cc.	Loss Iodine Percentage
Oct. 15, 1926.					
5	0	25	0	6.75	0
5	5	25	2 min.	5.00	26
5	5	25	15 min.	3.50	48
5	5	25	30 min.	3.00	55
Tetraiodomethenamine					
5	0	25	0	6.85	0
5	5	25	2 min.	5.90	14
5	5	25	15 min.	4.40	36
5	5	25	30 min.	4.05	41
Oct. 20, 1926.					
Iodine					
5	0	50	0	6.55	0
5	5	0	20 min.	4.80	27
5	5	25	24 min.	4.35	33
5	5	100	29 min.	3.50	47
Tetraiodomethenamine					
5	0	50	0	6.75	0
5	5	0	15 min.	5.80	14
5	5	50	20 min.	4.90	27
5	5	100	24 min.	4.50	33
Oct. 27, 1926.					
Iodine					
5	0	50	0	6.50	0
5	5	50	0.5 hrs.	3.90	40
5	5	50	2.0 hrs.	2.85	56
5	5	50	2.75 hrs.	2.85	56
5	5	50	23.0 hrs.	1.20	82
5	5	50	47.0 hrs.	0.65	92
Tetraiodomethenamine					
5	0	50	0	6.65	0
5	5	50	0.5 hrs.	4.45	33
5	5	50	2.0 hrs.	3.60	46
5	5	50	2.75 hrs.	3.60	46
5	5	50	23.0 hrs.	1.80	73
5	5	50	47.0 hrs.	1.10	83

skin appeared to be healthy. Recently, in view of a consideration of our experimental data, we changed our technic to include a preliminary application to the lesions of some talc powder containing 10 per cent potassium iodide. This not only served to direct the iodine out of the membrane toward the skin rather than away from it, but also gave us on the skin the equivalent of solution 3 in tables 1 and 2 (potassium iodide plus methenamine), which mixture appears to have fungistatic action. It must be understood that as iodine leaves the membrane, it releases methenamine, which also leaves the membrane, as it has a

greater solubility in water (visible and invisible perspiration and vesicular fluids) than in collodion.

Our experience with this form of therapy is limited to its use on patients in private practice. A summary of this experience emphasized the following points:

1. It is useless to attempt to treat patients who have the deep vesicular or markedly exudative types of dermatophytosis by this method without the preliminary use of an effective keratolytic and of an agent to dry up the exudation, since in the former condition the membrane and its contained drugs cannot get at the fungi, and in the latter condition the membrane tends to lock in the secretions; thereby producing pain and swelling.

2. Itching is readily relieved by the application of collodion.

3. The membrane gives a dry, clean dressing for which the patients are thankful.

4. Lesions that had persisted for two years in spite of the use of salves and roentgen-ray treatment cleared up within fourteen days.

5. Recurrence was the rule. This statement, however, must be judged in the light of an experimental technic applied in a condition in which cures can only be guessed at by the appearance of the skin. We have not as yet made an attempt to use this compound in any excipient but collodion. Because of a happy experience in the one attempt at treating tinea of the scalp in a child, we may be permitted to suggest that tetraiodomethenamine in water solution might with reason be effectively used as a prophylactic against the epidemic spread of ringworm of the scalp in institutions for children. The solution may be applied daily to the scalp in fungicidal strength without risk of irritation of the skin.

We wish to express our thanks to Dr. Howard Fox and Miss Avchen of his laboratory for performing the experimental work with fungi herein reported.

DISCUSSION

DR. F. D. WEIDMAN, Philadelphia: We should attempt to cure by killing the organism. Clinical experience has shown that many patients who appear to be cured have a recurrence; that is the complaint not only of the specialist, but also of the general practitioner. The hygiene of the problem is so complicated that I doubt whether the difficulty can be met by that route. It is impossible to sterilize all of the wearing apparel of the patient; even so, there would still remain the floor coverings in our homes, hotels, shower baths, etc. It seems to me that the real approach to the millenium in the way of treatment is to establish conditions in the intertriginous locations, in which the fungi work and mobilize, which will make these locations undesirable to the fungus. In the meantime, it appears that we shall have to worry along with the parasiticides, and whereas we have some that are satisfactory, we welcome others such as those that have been brought to our attention by the essayists.

DR. T. CASPAR GILCHRIST, Baltimore: I think there is an epidemic following the war. We are astonished that so many common diseases, such as eczema,

pomphlox and others, are probably due to fungus infection. Many of them were formerly considered due to internal causes, but are now known to be due to ringworm infection; we shall have many remedies that will help us out in these external rather than internal diseases. There are infections between the fingers and toes, which extend up the arms and legs, and which are due to the ringworm fungus. This was described by Sabouraud in 1904, and many variations have been described since that time. Patients are cured, but come back later in as bad condition as before. Why? Because they have been reinfected, often by the use of golf clubs, umbrellas, clothing and other things. As Dr. Weidman said, we cannot burn down the house and boil the patient, and I think all physicians have a different remedy for ringworm. I wish to mention the vesicular form of ringworm. When mercurochrome-220 soluble is applied in these cases, it has a stimulating effect and makes the eruption spread more rapidly.

Dr. Harold N. Cole, Cleveland: I should like to ask Drs. Highman and Sharlit whether their preparation is the same as that known on the market under the name of cyanamide.

Dr. Howard J. Parkhurst, Toledo, Ohio: I wonder whether any one has found that thymol and oil of cinnamon are often irritating when applied in the recommended strengths of 5 per cent and 2 per cent, respectively. I have had that experience in my cases, especially with the oil of cinnamon, which I have finally abandoned. I find that the strength of the thymol solution often has to be reduced, and sometimes I have had to discard the use of this oil entirely.

Dr. Everett S. Lain, Oklahoma City, Okla.: This subject is one which interests every dermatologist. Even general practitioners are reading about the subject and calling on the dermatologists for more information, and are treating patients with these infections with more or less success. The resistance and frequency of the disease seem to vary somewhat with climate. I have recently reviewed more than 2,300 dermatologic cases of patients who came to our office during 1926; I found that more than 200 were cases of infections; about one third were proved microscopically. We do not make routine microscopic examination in every case, since the clinical manifestations are so obvious in many. In our practice, the epidermophyton infections are about 10 per cent of the total number of cases examined. We know that this organism grows best on the covered parts of the body where moisture and heat are more in evidence; also conducive to its growth is friction between the interdigital surfaces and in the inguinal region. In the treatment we have tried everything recommended. In 1925, after the recommendation of aromatic oils, we gave this treatment a fair trial, but we had the same trouble mentioned by Dr. Parkhurst; namely, extreme irritation, which caused us to abandon treatment with aromatic oil and thymol. We have fallen back on the treatment we had previously used for several years and have found it the most satisfactory; namely, salicylic acid, about 1 drachm (2.5 minims) to 1 ounce (28.35 Gm.) of 70 per cent alcohol. We apply this solution for a few days to produce rapid exfoliations, then we usually follow with Whitfield's ointment. When this is applied, we place gauze between the toes and fingers to prevent friction. This dressing has become a routine treatment in our office.

Dr. F. J. Eichenlaub, Washington, D. C.: Of 800 patients examined as a routine, about 43 per cent presented clinical evidence of ringworm infection, chiefly the interdigital type on the feet. Of this 43 per cent, about 25 per cent of the cases were complicated by ringworm elsewhere, such as tinea cruris and ringworm eczema. Regarding the use of mercurochrome-220 soluble the laity

and the physicians in Washington use this commonly, and many of our patients have used the drug for a long time before consulting us. In my experience, it has been of little value.

Dr. Moses Scholtz, Los Angeles, Calif.: I am not qualified to discuss the subject from a bacteriologic point of view, but, irrespective of the clinical virtues of the suggested remedy, as a clinician I would express an opinion that the clinical problem of dermatophytosis will not be solved by antiseptic and antimycotic specific drugs. It seems to me that the chemicals and formulas at our command at present, such as Whitfield's ointment, thymol and cinnamon and many others, are practically specific in their antiseptic clinical effect on accessible fungi. The clinical stubbornness and therapeutic resistance of epidermophytosis, in my opinion, is due essentially to latent potentialities and opportunities for reinfection and recurrent self-inoculation on the part of the infected person.

Until the clinicians direct their major efforts in this direction and institute a prophylactic and social educational campaign along the same lines, the clinical problem of epidermophytosis will continue to be unsatisfactory, in spite of the highest degree of efficiency of antiseptic local applications.

The second speaker has honored me by quoting extensively my recent paper on clinical diagnosis of epidermophytosis, for the purpose of refuting the validity of its deductions. My paper was intended for the general practitioner, to give him aid and guidance in the clinical recognition of epidermophytosis. Far be it from me to decry the desirability of bacteriologic investigation, but I believe that clinical experience overwhelmingly demonstrates the possibility of diagnosing and handling a large majority of cases of epidermophytosis by clinical data alone. The clinical incidence and dissemination of epidermophytosis is so tremendous that it is safe to say that only in a small percentage of cases is a thorough bacteriologic examination feasible or possible. This undeniable fact renders justifiable, desirable and necessary the portrayal of clinical differential diagnostic traits and earmarks of epidermophytosis such as those offered in my quoted paper.

Dr. Joseph Müller, Holden, Mass.: I wish to take exception to Dr. Eichenlaub's statement that mercurochrome-220 soluble is of little value in these cases. Dr. White has used it most successfully and almost exclusively for some time. I started it at about the same time, and I have found mercurochrome to be more satisfactory than anything else. It was absolutely useless if I gave a bottle to the patient and allowed him to use it whenever he wanted to; but in the hands of the dermatologist who will not use it too frequently, I think it is more valuable than the other remedies. It is not irritating like iodine and the volatile oils, and seems to have a good action by exterminating the parasite. In some cases I found it necessary to clean and sterilize everything around the patient; it was necessary to have him change his clothes and shoes and to have all the floors thoroughly disinfected.

Dr. Walter J. Highman: I will request Dr. Sharlit to answer Dr. Cole's question, for I do not know the names of these things. It was not Dr. Sharlit's intention or mine to suggest a method of treatment that offered a final solution of the ringworm problem, but simply to offer one more line of attack when other methods had proved unsatisfactory. I would not say that the results of our investigations were in any way brillant except in the superficial cases; in these cases, of course, when the free, nascent iodine was able to reach the offending organisms, it was possible to destroy them. I avoid the word "cure" for it is bandied about too promiscuously.

The great difficulty in the treatment of patients who have tinea of the skin arises from the depth of the lesions, and unless they can be converted from deep into superficial lesions, this treatment, as all other, would be a failure. One of the ways that we have found efficacious in emptying these gesicles is to give a herterotonic salt bath to the patient twice a day. The treatment outlined can be combined with other therapeutic agents or varied with other forms of therapy. Any antiseptic treatment consistently and intelligently applied, will always be of value in some cases, but there will be a certain percentage of patients that will not respond to any treatment, and it is for those cases that we present the new prospect.

Dr. Herman Sharlit, New York: Replying to Dr. Cole, the preparation is made of a combination of iodine and methenamine. The tablet Dr. Cole asked about, contains, among other things, this preparation. It is given in an attempt to supply the necessary iodine.

EXCRETION OF SUGAR IN SWEAT

ITS RELATIONSHIP TO ECZEMA *

B. USHER, M.D.
AND
I. M. RABINOWITCH, M.D.
MONTREAL

Within recent years, observations have been recorded in the literature which suggest a relationship between eczema and disturbed metabolism of carbohydrates.[1] In some patients who have both diminished tolerance for sugar and eczema, it has been noted that the latter condition has cleared up rapidly following the institution of diets of low carbohydrate content. That skin conditions may be related to the metabolism of sugar is further suggested by the striking manner in which the pruritus pudendi of diabetes vanishes after the disappearance of sugar from the urine. Whether the underlying disturbance is local (action of sugar on the skin and mucous membrane) or general (metabolic) in this condition, is still problematic. In view of these observations, an attempt was made to determine whether there was a relationship between the percentage of sugar found in sweat and eczema.

Little is known about the sugar content of sweat. As far as could be ascertained, the references in the literature are extremely scanty. Talbert [2] made an examination of the secretions of ten normal subjects, and states that he found sugar in all. The report is brief, and details as to technic, etc., are not given. The statement that sugar is a constituent of sweat is found in various textbooks. These, however, record observations made on patients suffering from diabetes; as far as could be ascertained, mention was not made of nondiabetic persons. An investigation was made, therefore, with three objects in view, namely: (*a*) to determine, by modern technic, whether sweat is a channel for the excretion of sugar in the normal person; (*b*) if it is, whether there are significant variations from the normal in persons with eczema, and if so (*c*) whether there are significant differences between persons with and without disturbances in the metabolism of carbohydrates.

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* From the Departments of Dermatology and Metabolism, The Montreal General Hospital.

1. McGlasson, I. L.: Hyperglycaemia as an Aetiological Factor in Certain Dermatoses, Arch. Dermat. & Syph. **8**:665 (June) 1920; Hyperglycaemia, ibid. **13**:338 (March) 1926. Ayers, S., Jr.: Glucose Tolerance Reactions in Eczema, ibid. **11**:623 (May) 1925. Campbell, G. G., and Burgess, J. F.: Intolerance to Sugar as a Factor in the Production of Some Dermatoses, Brit. J. Dermat. **39**:187, 1927. Rabinowitch, I. M.: Blood Sugar Time Curves, J. Clin. Investigation **2**:579, 1926.

2. Talbert, G. A.: Physiology of Sweat, J. A. M. A. **87**:777 (Sept. 4) 1926.

METHOD OF INVESTIGATION

The subjects were patients selected at random as they entered the outdoor dermatologic clinic of the Montreal General Hospital. Among these were persons with and without eczema. The conditions of the subjects who did not have eczema included acne vulgaris, syphilis, verrucae and seborrheic dermatitis of the scalp. All subjects were males.

Collection of Material.—The procedure for the collection of sweat was that described by Barney,[3] with but slight modifications. All materials were collected in the morning and analyzed immediately. In each case, the subject was not allowed food after 7 o'clock the evening before the test, at which time a cleansing bath was ordered. As the subjects reached the laboratory, each patient was placed between rubber sheets, and allowed to remain there for one hour. To produce sweating, two hot water bottles were placed at either side of the patient, after which he was generously covered with blankets. As some of these persons were to receive dextrose by mouth and as their blood was to be analyzed, arrangements were made so that maximum secretion would take place approximately at the time one expects the maximum concentration of sugar in the blood following the ingestion of dextrose. As the maximum concentrations of sugar in the blood are usually noted about thirty minutes after the ingestion of dextrose and as maximum secretion of sweat usually is noted fifteen minutes following the administration of pilocarpine, a routine procedure was adopted for comparative purposes, whereby all patients received subcutaneous injections of one-fourth grain (0.016 Gm.) pilocarpine fifteen minutes after having been placed between the rubber sheets.

The subjects were divided into two groups: (*a*) those who were given sugar and (*b*) those who were given only water. In each case, 250 cc. of fluids were given before and one-half hour after the test had started.

All tests commenced while the patients were in the postabsorptive (fasting) state. From some of the subjects who received only water, blood was collected for analysis before and one-hour after the test, and from others only before the test. From the subjects receiving dextrose, blood was collected before, one-half hour and one hour after ingestion of 100 Gm. of dextrose.

The specimens of sweat were, as was to be expected, slightly turbid, because of the admixture of epithelial debris, etc. In order to remove the latter the materials were cleared by means of kaolin. For control purposes, several samples of sweat were examined for their sugar content both before and after treatment with kaolin. Appreciable differences were not noted.

Method of Analysis.—For the determination of sugar in the blood, the method of Folin and Wu was applied with slight modification. Dilution was found unnecessary for the precipitation of the proteins; this was obtained by adding one volume of 10 per cent sodium tungstate to one volume of sweat, and then adding drop by drop one volume of 0.66 normal sulphuric acid. After the mixture was then shaken for fifteen minutes and filtered, a clear filtrate was obtained. The further details of the technic were the same as for blood, except that, since the amount of reduction of copper was small, a more dilute sugar standard was used.

In all the samples of sweat examined, a definite reduction of copper was found. In order to ascertain whether these reductions were due to dextrose, the following procedure was carried out:

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3. Barney, R. E.: Chemical Analysis of Sweat, J. A. M. A. **85**:1373 (Oct. 31) 1925.

About 1 Gm. of fresh yeast was placed in a 25 cc. Folin-Wu blood sugar tube; to this some sweat was added. The mixture was well shaken, and more sweat was added to the 25 cc. mark. A uniform mixture was obtained by inverting the tube a few times. In another tube a like amount of yeast was treated similarly with a solution of dextrose (1 cc. equals 0.1 mg.). A third tube contained yeast and distilled water, and a fourth tube sweat only. Thus: tube 1, yeast plus sweat; tube 2, yeast plus solution of glucose; tube 3, yeast plus distilled water; tube 4, sweat only. To each tube was added a few drops of toluene as a preservative. All four tubes were placed in an incubator at body temperature for twenty-four hours. As a further precaution, in order to control the effects of bacterial activity, a fifth tube containing sweat and toluene was kept in a refrigerator during the same period of time. It was found that unless the control procedure described was followed, utterly unreliable data were obtained. After twenty-four hours, all tubes were examined to determine their sugar content after distilled water had been added to the 25 cc. mark wherever it was necessary in order to correct for evaporation.

Tubes 1, 2 and 3, that is, those containing yeast, did not show any reductions, while tube 4, which contained only sweat, yielded, within the limits of experimental error, the same reductions as those found in the specimens immediately on collection and in that which was kept on ice as a control.

It may be stated that as a still further control, distilled water was allowed to remain in the rubber sheets for one hour, and its reducing properties were then tested. Reducing substances were not found in the water following this procedure.

In view of the method of control, it appears reasonable to assume that the reductions obtained were due to the presence of a fermentable substance, which was probably dextrose.

In order to determine whether there were appreciable differences between the secretions of persons with and without eczema or between those with and without disturbances of metabolism for carbohydrates, eleven normal subjects were first studied; the data are recorded in tables 1 and 2. In six cases, analyses of sweat were made with the subject in the fasting state (table 1), and in five instances data were obtained following ingestion of dextrose (table 2).

It will be noted that there were marked variations in the amounts of sweat and also in the sugar values, both as to concentration and as to the rate of excretion. Little information was obtained without further analysis. A series of abnormal cases were collected; all of these cases were then classified as follows:

Group 1.—Subjects with eczema.
(*a*) Normal metabolism of carbohydrates.
(*b*) Diminished tolerance of carbohydrates.

Group 2.—Subjects without eczema.
(*a*) Normal metabolism of carbohydrates.
(*b*) Diminished tolerance of carbohydrates.

Sweat for analysis for sugar was obtained from each group, both in the fasting state and following ingestion of 100 Gm. of dextrose. The results obtained are recorded in detail in tables 3 to 7.

TABLE 1.—*Results of Analysis of Sweat Obtained During Fasting State from Normal Persons*

No.	Blood Sugar (per Cent)	Sweat		
		Amount (Cc.)	Sugar	
			Mg. per 100 Cc.	Mg. per Hour
1	0.113	150	26	39
2	0.106	150	16	24
3	0.094	175	13	23
4	0.106	40	27	11
5	0.104	75	10	8
6	0.108	30	14	4
Average		113	18	18

TABLE 2.—*Results of Analysis of Sweat Obtained Following Ingestion of Dextrose by Normal Persons*

No.	Blood Sugar (per Cent)			Sweat		
	Before Dextrose	½ Hr. After	1 Hr. After	Amount (Cc.)	Sugar	
					Mg. per 100 Cc.	Mg. per Hour
1	0.108	0.133	0.130	70	14	10
2	0.120	0.180	0.176	100	22	22
3	0.090	0.140	0.142	200	14	28
4	0.098	0.138	0.153	65	22	14
5	0.110	0.151	0.162	100	18	18
Average				147	18	18

TABLE 3.—*Results of Analysis of Sweat Obtained During Fasting State from Patients with Eczema and with Normal Tolerance for Sugar*

No.	Blood Sugar (per Cent)	Sweat		
		Amount (Cc.)	Sugar	
			Mg. per 100 Cc.	Mg. per Hour
1	0.103	100	21	21
2	0.098	25	24	6
3	0.091	60	12	7
4	0.100	50	17	9
5	0.096	100	21	21
Average		67	19	13

TABLE 4.—*Results of Analysis of Sweat Obtained Following Ingestion of Dextrose from Patients with Eczema and Normal Tolerance for Sugar*

No.	Blood Sugar (per Cent)			Sweat		
	Before Dextrose	½ Hr. After	1 Hr. After	Amount (Cc.)	Sugar	
					Mg. per 100 Cc.	Mg. per Hour
1	0.103	0.109	0.070	125	14	18
2	0.095	0.137	0.139	200	14	28
3	0.091	0.123	0.112	25	24	6
4	0.083	0.166	0.147	200	14	28
5	0.100	0.140	0.151	65	22	14
6	0.128	0.180	0.178	100	22	22
7	0.098	0.112	0.082	125	19	24
8	0.096	0.156	0.152	90	15	14
Average				116	18	19

Tables 3 and 4 contain data concerning patients with eczema but with normal metabolism of carbohydrates; in table 3 are the data obtained while the patient was in the fasting state, and in table 4 are those obtained following the ingestion of dextrose. Tables 5 and 6 contain data concerning patients with eczema but with diminished tolerance for carbohydrates; in table 5 are the data obtained from persons in the fasting state and in table 6 are those obtained from subjects following the ingestion of dextrose.

TABLE 5.—*Results of Analysis of Sweat Obtained During Fasting State from Patients with Eczema and Diminished Tolerance for Sugar*

		Sweat		
			Sugar	
No.	Blood Sugar (per Cent)	Amount (Cc.)	Mg. per 100 Cc.	Mg. per Hour
1	0.107	50	17	9
2	0.119	125	14	18
3	0.122	300	12	36
4	0.122	50	5	3
5	0.122	20	28	6
Average		109	15	14

TABLE 6.—*Analysis of Sweat Obtained Following Ingestion of Dextrose from Patients with Eczema and Diminished Tolerance for Sugar*

	Blood Sugar (per Cent)			Sweat		
					Sugar	
No.	Before Glucose	½ Hr. After	1 Hr. After	Amount (Cc.)	Mg. per 100 Cc.	Mg. per Hour
1	0.125	0.164	0.263	50	18	9
2	0.119	0.200	0.181	200	17	34
3	0.122	0.212	0.222	800	4	32
4	0.096	0.212	0.200	125	15	19
5	0.123	0.212	0.185	500	15	75
Average				338	14	34

RESULTS

In spite of the wide fluctuations noted in all groups of cases, it will be seen that the average concentrations of dextrose (expressed as milligrams per hundred cubic centimeters) show relatively little variation and appear to be independent of the conditions under which the specimens of sweat were collected, i. e., whether the specimens were collected while the subject was in the fasting state or after he had ingested dextrose. The concentrations ranged from 14 to 19 mg. When, however, the average rates of excretion (expressed in terms of milligrams per hour) are considered, one striking difference is noted: in the fasting state hardly any differences are found, the concentrations ranging from 13 to 19 mg. Following the ingestion of dextrose, however,

both the patients with eczema and those with diminished tolerance for sugar showed an increase in the average rate of the excretion of sugar, namely, 34 mg. It will be noted that in the fasting state, the average rate of excretion of this same type of subject was 14 mg. per hour, which was the same as for the other subjects. It will be noted, therefore, that in this type of case the essential difference was in the volume

TABLE 7.—*Sweat Obtained During Fasting State and Following Ingestion of Dextrose from the Same Normal Person on Different Days*

		Blood Sugar (per Cent)			Sweat		
						Sugar	
Subject	Condition	Before Dextrose	½ Hr. After	1 Hr. After	Amount (Cc.)	Mg. per 100 Cc.	Mg. per Hour
1	Fasting	0.103	.....	.....	100	21	21
	After ingestion of dextrose	0.103	0.109	0.070	125	14	18
2	Fasting	0.096	.....	.....	25	24	6
	After ingestion of dextrose	0.096	0.156	0.152	90	15	14
3	Fasting	0.082	.....	.....	50	17	9
	After ingestion of dextrose	0.082	0.162	0.160	45	17	8
4	Fasting	0.092	.....	.....	100	21	21
	After ingestion of dextrose	0.092	0.140	0.152	100	20	20
	Average—Fasting				69	21	14
	After ingestion of dextrose				90	17	15

TABLE 8.—*Analysis of Sweat Obtained During Fasting State and Following Ingestion of Dextrose from the Same Person with Lowered Tolerance for Sugar on Different Days*

		Blood Sugar (per Cent)			Sweat		
						Sugar	
Subject	Condition	Before Dextrose	½ Hr. After	1 Hr. After	Amount (Cc.)	Mg. per 100 Cc.	Mg. per Hour
1	Fasting	0.107	.....	.....	50	17	9
	After ingestion of dextrose	0.096	0.212	0.200	125	15	18
2	Fasting	0.119	.....	.....	125	14	18
	After ingestion of dextrose	0.119	0.200	0.181	200	17	34
3	Fasting	0.122	.....	.....	50	5	3
	After ingestion of dextrose	0.122	0.212	0.222	800	4	32
4	Fasting	0.122	.....	.....	300	12	36
	After ingestion of dextrose	0.123	0.212	0.185	500	15	75
	Average—Fasting				131	12	17
	After ingestion of dextrose				406	13	40

of sweat excreted. After the ingestion of glucose, there was an average of 335 cc. of sweat, whereas the quantities obtained under all other conditions were much smaller, varying between 67 and 115 cc. per hour. It would therefore appear that in patients with eczema accompanied by diminished tolerance for sugar there is an increase in the rate of excretion of sugar owing to an increase in the secretion of sweat.

In order to determine whether the difference was accidental or real, the problem was approached from another angle. Subjects with eczema were divided into those with normal and those with abnormal metab-

olism of carbohydrate. Sweat was collected on two different days: on one day while the subject was in the fasting state and on another following the ingestion of dextrose. The observations of four patients with normal metabolism of carbohydrates are recorded in table 7, and data concerning patients with disturbed metabolism of carbohydrates are given in table 8.

It will be noted that the subjects with normal metabolism of carbohydrates showed practically no disturbance in the rates of excretion of dextrose after its ingestion; the average rate was 14 mg. per hour when fasting and 15 mg. per hour when dextrose was given. Strikingly different results were obtained, however, when the patients showed a disturbance in the metabolism of carbohydrates. In the fasting state the excretions of sugar averaged 17 mg. per hour, whereas after ingestion of dextrose it was 40 mg. per hour. It is interesting to note that here also the volume of secretion was greater following the ingestion of dextrose in the cases in which the intolerance for sugar was lowered.

The foregoing observations lend themselves to certain speculations as to the relationship between eczema and increased excretion of sugar in sweat. It would appear from our observations and from the results of dietetic treatment reported by others that if dextrose is the exciting agent in these cases, the institution of diets low in carbohydrate content would lead to excretions of sugar and sweat which approach those of persons with a normal metabolism of carbohydrates. This view is strengthened by the recent observations of Weidman,[4] who reported that in two persistent "fungus negative" cases he succeeded in getting positive results (growth of fungus) by applying dextrose solution to the affected part for a period of a week. On the other hand, one of us (B. U.) has repeatedly observed the effects of intradermal injections of solutions of dextrose (from 0.1 to 1 mg. per cubic centimeter). The results were invariably negative. The experiments are not, however, strictly comparable. This phase of the subject is now being investigated.

SUMMARY

A fermentable reducing substance, assumed to be dextrose, because of the procedure followed, was found to be a normal constituent of sweat, and the rate of its excretion was studied.

The rate was found to be increased in cases of eczema in which the tolerance for sugar was lowered.

The volume of sweat has also been found to be increased in such cases.

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4. Weidman, F. D.: Laboratory Aspects of Epidermophytosis, Arch. Dermat. & Syph. **15**:415 (April) 1927.

A causal relationship between the excretion of sugar in sweat and eczema is suggested.

NOTE.—Since this paper was submitted for publication, an article appeared on "A Study of the Skin in 500 Cases of Diabetes" (J. A. M. A. **89**:774 [Sept. 3] 1927). It is interesting to note that the concentrations of sugar in sweat recorded were practically identical with our own observations, namely, 15 mg. per hundred cubic centimeters.

FUNGI AND FUNGOUS DISEASES

LECTURE I*

ALDO CASTELLANI, C.M.G., M.D.

Fellow of the Royal College of Physicians, London; Professor of Tropical Medicine, Tulane University, New Orleans; Visiting Lecturer, London School of Hygiene and Tropical Medicine and University of Porto Rico; Director of Tropical Medicine, Ross Institute, London; Emeritus Professor of Tropical Medicine, Ceylon Medical School

NEW ORLEANS

(Continued from page 604)

CLASS III. PHYCOMYCETES

The *Phycomycetes,* which are characterized by the mycelium being continuous and nonseptate, in the vegetative stage, contain several families, one of which, *Mucoraceae,* has organisms parasitic of man. The fungi belonging to this family have a branching mycelium with aerial branches (gonidiophores), each of which suports on its distal extremity a globular, pear-shaped or claviform sporangium, called gonidangium, which is at first separated from the gonidophore by a septum. This septum later protrudes into the lower portion of the sporangium and forms a variously shaped structure known as columella. By free cell formation, gonidia develop inside the sporangium. The sporangial protoplasm not used in the formation of the gonidia is transformed into a peculiar mucillaginous substance, which later dries up and is the cause of the sporangium bursting. Each gonidium which becomes free gives rise by germination to a mycelial filament. There is also at times a sexual form of reproduction which consists in the conjugation of undifferentiated nonmotile gametes, with the formation of zygospores. When growing in unfavorable mediums numerous species reproduce only by conidia and chlamydospores.

The family *Mucoraceae* contains a number of genera, among which the following have species parasitic of man:

1. *Mucor* Micheli, 1729. Mycelium ramified, rhizoids absent.

2. *Rhizomucor* Lucet and Costantin, 1900. Rhizoids (slender rootlike filaments) present, columella ovoid.

3. *Rhizopus* Ehrenberg, 1820. Rhizoids present, columella mushroom-like.

4. *Lichtheimia* Vuillemin, 1904. Peduncle supporting sporangium ends in a special formation encircling the base of the columella.

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*The Adolph Gehrmann Lectures of the University of Illinois College of Medicine, 1926.

For a more complete classification of the *Phycomycetes,* the reader is referred to the "Manual of Tropical Medicine" by Castellani and Chalmers.[13]

SIMPLE CLASSIFICATION OF FILAMENTOUS FUNGI INCLUDING MOLDS FOR THE USE OF CLINICIANS AND CLINICAL PATHOLOGISTS

I will present here a simple scheme for the classification of filamentous fungi, just as I did in the case of so-called yeastlike fungi.

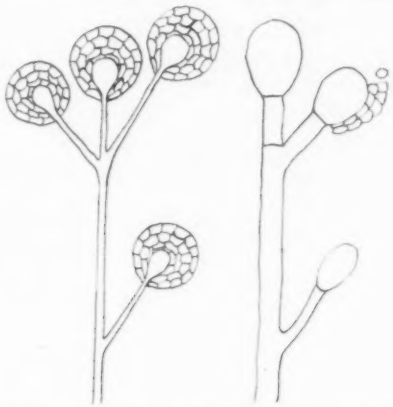

Fig. 36.—Rhizomucor parasiticus Lucet and Costantin.

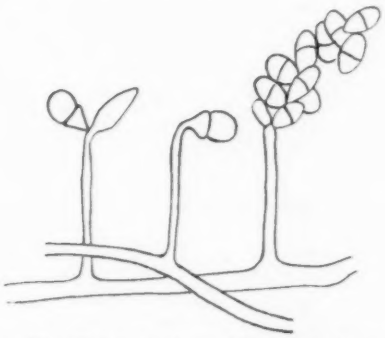

Fig. 37.—Trichotecium (from Buchanan).

Filamentous fungi, viz., fungi with abundant mycelial filaments, while free yeastlike or budding cells are scanty or absent, may from a practical point of view, be separated into two large groups:

Group A: well defined fructifications absent, cultures usually not fluffy, *Nocardia-oidium* group *sensu lato.*

Group B: well defined fructifications or specially shaped conidia present, cultures often fluffy, molds group *sensu lato.*

13. Castellani and Chalmers (footnote 3, p. 972).

Group A may be divided into two subgroups:

1. Mycelial threads thin, bacillus-like—*Nocardia sensu lato* (comprises *Nocardia, Cohnistreptothrix, Leptothrix, Vibriothrix.*
2. Mycelial threads of larger dimensions, often closely septated—*Oidium sensu lato* (comprises *Oidium sensu stricto, Trichosporum, Madurella, Indiella hemispora*).

Group B, which is characterized by mycelial threads of a fairly large size, cultures that are often fluffy and the presence frequently of distinctive fructifications, comprises those fungi which in clinical laboratories are known as molds. It comprises also the *Trichophyton, Microsporum, Achorion, Epidermophyton, Endodermophyton* group of

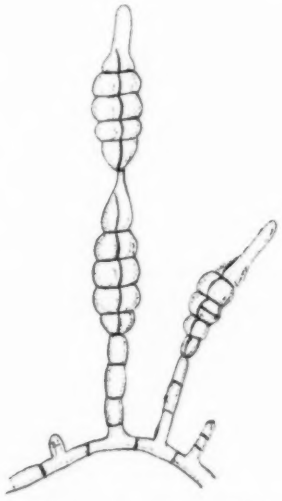

Fig. 38.—Alternaria (from Buchanan).

fungi, the cultures of which, especially when old, have some similarity to ordinary molds.

Group B (molds *sensu lato*) may be classified as follows:

1. Isolated from ringworm or ringworm-like conditions; large spindle-like terminal structures (fuseaux) often found in cultures—*Trichophyton sensu lato* (comprises *Trichophyton, Microsporum, Epidermophyton, Achorion* and *Endodermophyton*).
2. Often *Saprophytes,* and when pathogenic do not as a rule cause ringworm-like lesions; fuseau absent; distinctive fructifications often ringworm-like lesions; fuseaux absent; distinctive fructifications often present—molds *sensu stricto.*

The fungi that are known as molds in clinical laboratories (*Aspergillus, Penicillium, Mucor,* etc.) belong to different classes of fungi—*Ascomycetes, Phycomycetes,* etc., of which I have given a botanic

description in the preceding pages. For practical purposes they may be described as characterized by a much branched mycelium and growing on ordinary laboratory mediums and also on decaying material, as a cottony, fluffy or velvety mass.

Buchanan gives the following useful key, based on the characters of the conidia (asexual spores), for the recognition of the nine most common genera.

Key for Differentiation of Common Genera of Molds

A. Spores borne in sporangia. Mycelium nonseptate.

B. Sporangiophores arising in clusters from the nodes of runners or stolons.....1. *Rhizopus*

BB. Sporangiophores arising singly, without stolons..2. *Mucor*

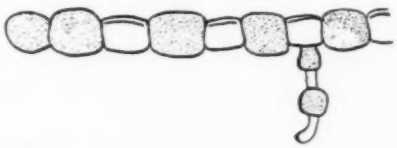

Fig. 39.—Chlamydospores of Mucor.

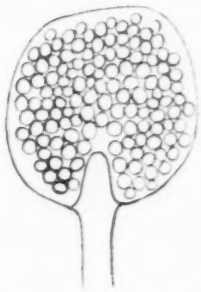

Fig. 40.—Sporangium of Mucor.

AA. Spores (conidia) never borne in sporangia.

B. Neither conidia nor hyphae smoky or dark in color.

C. Conidia 1 celled.

D. No distinct conidiophores. Conidia formed as oidia.....3. *Oöspora*

DD. Conidia on distinct conidiophores.

E. Conidiophores enlarged or club-shaped at tip.....4. *Aspergillus*

EE. Conidiophores branched at tip....5. *Penicillium*

CC. Conidia two-celled, pear-shaped .....6. *Trichothecium*

BB. Either conidia or hyphae smoky or dark, or both.

C. Conidia one-celled, in branched chain.....7. *Hormodendrum*

CC. Conidia more than one-celled.

D. Like *Hormodendrum*, but conidia two-celled when old.....8. *Cladosporium*

DD. Spores many-celled, club-shaped, in chains....9. *Alternaria*

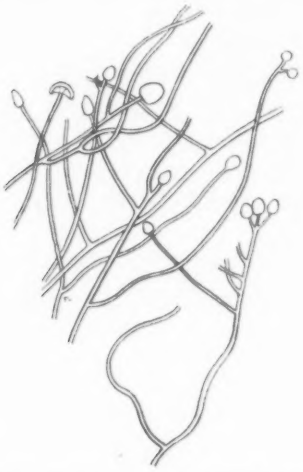

Fig. 41.—Rhizomucor septatus Von Bezold (after Siebemann).

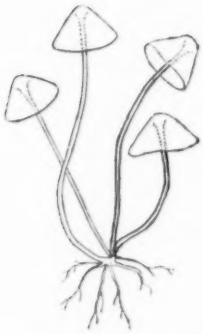

Fig. 42.—Rhizopus niger Ciaglinski and Hewelke.

BIOLOGIC CHARACTERS OF FUNGI

I will now pass from the morphologic characters of fungi to a more interesting subject: their biologic and biochemical characteristics. The more one studies the fungi, the more one sees how closely related they are to true bacteria with regard to biologic and biochemical characters.

Agglutinins.—Twenty-five years ago the late Dr. Macfayden, director of the Lister Institute, inoculated rabbits with cultures of a saccharomyces, and observed a production of specific agglutinins in the

blood for the particular species he had inoculated. These results were confirmed and enlarged, using other fungi, by G. H. Rogers, Concetti, Quarelli and others, and Widal and Abrami have described a general diagnostic method, "Sporo-agglutination Method," based on the observation that in patients suffering from mycologic diseases are found specific agglutinins for the spores of the causative fungi. In my experience, however, in many cases, in addition to specific agglutinins, there is present also a large amount of nonspecific agglutinins, and this generally detracts from the usefulness of the method.

Toxins.—Fungi may produce powerful toxins. Some years ago, Auchair and Verliac obtained a toxic substance soluble in ether from *Nocardia bovis,* and they called it "Actinomycetine." Charrier, Roger and others have succeeded in extracting toxins from monilia fungi. Various toxins have also been extracted from fungi of the genus *Aspergillus,* and at one time a number of authorities, including Ceni, Besta and others, believed that pellagra was caused by these aspergillus toxins.

Vaccines.—Mycetic vaccines have been prepared by various authorities. Plato and others prepared a trichophyton and microsporal vaccine by killing cultures of different trichophytons and microsporons by heat, and then triturating the mass. By using vaccines intradermally various cutireactions have been devised; for instance, De Beurmann devised a sporotricho-cutireaction for the diagnosis of sporotrichosis; others have devised a trichophitic cutireaction for the diagnosis of atypical cases of ringworm, etc.

Biochemical Changes Induced by Fungi.—Certain fungi may induce profound chemical changes and play an extremely important rôle in nature and are of great importance in agriculture and industry. Certain fungi are intimately connected with the metabolism, life history and successful development of the higher plants. Mycelial fungi grow on and in the roots of a large number of these plants, and it is probable that in some cases nitrogen is furnished to the growing plants by these organisms. The rôle played by organisms of the family *Saccharomycetaceae* with regard to alcoholic fermentation, and the production of wine, beer, cider and other fermented beverages, is well known.

Fungi are of great utility in the ripening of cheese, and the flavor of each particular cheese is to a great extent due to the different fungi present. For instance, *Penicillium roquefortii* gives the characteristic taste of Roquefort cheese. The odor and, to a great extent, the taste of Camembert cheese is caused by *Penicillium camembertii,* in addition to other molds, such as *Oidium lactis.*

SYMBIOSIS

A number of fungi are capable of living in symbiosis. The subject of symbiosis has so far attracted little attention from medical men. I use of the term symbiosis *sensu lato* to indicate the living together of two organisms in close association, their close association not being detrimental to either of them; symbiosis *sensu stricto* means that the association not only is not detrimental, but also is mutually beneficial. Botanists years ago discovered that a large group of plants—the lichens—represent a symbiosis of a fungus and an alga; they also found that symbiosis plays an important rôle in the nutrition process of numerous plants and in the germination of certain seeds. I shall deal briefly with symbiosis in general, as so little on the subject can be found in medical books.

Fig. 43.—Cryptococcus graciloides and Bacillus vermiculoides living in symbiosis.

Symbiosis Between Two Vegetal Organisms.—Composite Plants: The best known example is furnished by the lichens. A lichen is a composite plant consisting of a green alga and a fungus which may be grown separately with difficulty. Every one has observed the green dust that often occurs on lichens. This powdery dust consists of young associations called soredia of algal cells and fungal hyphae. Another well known symbiotic plant is the ginger-beer plant, the lumps of which are composed of a yeast (*Saccharomyces pyriformis*) and a bacterium (*Bacterium vermiforme*).

Symbiosis Between a Vegetal Organism and an Animal Organism.—Symbiosis may also occur between plant and animal. For instance, in the marine worm convoluta an alga is always present, and in the larvae of certain aphids, coccids, etc., yeasts occur as more or less definite structures.

Importance of Symbiosis in the Nutrition Processes of Certain Plants.—Reissek, in 1847, came to the conclusion that fungi were normally present within the cortical cells of the roots of various flowering plants, being best developed in the underground roots of orchids. Soon after, another type of association was noted, especially in forest trees. Here the fungus mycelium forms a sort of mantle around the root, in contrast to being within the cells of the cortex. This association is common in Castanea and Corylus. The rootlets so infected generally take a characteristic coralloid appearance. Frank, in 1885, coined the term "mycorrhiza" to denote fungi (including bacteria) in or around the roots of plants. He used the term "endotrophic mycorrhiza" for those forms in which the fungus occurred within the tissues of the host, and the term "ectotrophic mycorrhiza" when the fungal mycelium surrounded the rootlets as a sheath.

The antiquity of such associations is seen in the fact that they occur in fossil plants such as *Rhynia, Hornea* and *Asteroxylon* from the lower (or middle) Devonian period. Weiss, in 1906, recorded mycorrhiza in fossil roots from the lower coal measures, for which he suggested the name "mycorrhizonium."

A well known instance of symbiosis is represented by the nodules found in the roots of many leguminous plants. For many years it was noted that roots of leguminous plants, such as peas and clover, presented a large number of nodules or tubercles which were for a long time considered to be normal morphologic features of such plants. Later it was found that these nodules are formed by the presence of a bacillus (*B. radicola*). This bacillus is apparently always present in the soil; the bacillus enters through a root-hair, it reaches the interior of the cortex and begins multiplying there, producing the nodular outgrowths. The organism feeds and grows principally on the carbohydrates (sugar, etc.) supplied by the host plant, these having been produced as the result of the photosynthetic activity of plant leaves. The bacillus is therefore supplied by the plant with carbohydrate food, but the bacillus in return supplies the plant with the nitrogenous compounds necessary for the production of protoplasm, by utilizing the free nitrogen of the air circulating in the soil.

Most plants have to take their nitrogen in a combined form, as ammonia salts, nitrates, etc., as nitrogen is an inert element and difficult to force into combination with others. *B. radicola* is one of the few organisms which can perform this work, provided that it is supplied with the means of obtaining the energy required for the process in the form of appropriate carbohydate nutrition. According to most authorities, the bacillus will do the same thing when cultivated outside the plant under strict experimental conditions.

After the bacilli have thriven for a while, mainly at the expense of the food supplied by the root in which they are living and multiplying, many of the individuals become weaker and undergo a sort of degeneration, while a few pass into a resting stage, in which they become highly resistant to adverse conditions of life. The leguminous plant, which previously gave out carbohydrate food to the bacillus, then begins to receive: it receives from the degenerated mass of bacilli the stores of nitrogenous matter they have accumulated, and this is a good return for the sugars which it had previously expended. The comparatively few surviving bacilli serve to infect the soil, as the roots gradually rot, and then they are enabled to attack the roots of new leguminous plants.

Higher Plants.—The phenomena of symbiosis between root and fungi occur also in many plants higher than the leguminous, for instance, in forest trees. In these trees the fungus mycelium forms a sort of mantle round the root, in contrast to being inside the cells of the cortex. The association is common in Castanea and Corylus, the rootlets so infected generally taking a characteristic coralloid appearance.

Importance of Symbiosis in the Phenomena of Reproduction.—Germination of Seeds: Noel Bernard, in 1902, proved experimentally that certain seeds, as the seeds of orchids, can germinate only in the presence of appropriate fungi. If the seedling is not infected with the appropriate fungus, generally the same fungus which infects the roots of the adult parent plant, germination, as a rule, will not take place, or it will be slow and imperfect. Burgeff came to the same conclusion; viz., that orchid seeds germinate without difficulty when the appropriate fungus is supplied to them. It is interesting to note that before this was scientifically known, orchid growers had found it necessary to sow orchid seeds in the soil containing the parent plant in order to make them grow; the explanation is that the seed in this way became infected with the fungi necessary to induce germination, viz., the fungus found in the roots of orchids.

A few more details may be of interest: Orchid seeds taken from the capsule under sterile conditions and sown on ordinary substrata where fungus is not present do not, as a rule, develop. The only phenomenon observed in these conditions is that usually the seeds swell, and after two or three months—viz., after the reserve food, oil, part of which becomes transformed into starch, has been exhausted—they become green and soon after die, unless the fungus infestation takes place. At first it seems peculiar that the seeds should die after the production of chlorophyle: one would rather expect that autonomous growth by action of photosynthesis should take place; as T. Ramsbottom aptly put it, it appears that the seedling forms chlorophyle as a sort of a last despairing effort.

Importance of Symbiosis in Fermentation Phenomena.—Two organisms, neither of which alone produces gas-fermentation in certain

carbohydrates, may do so when living in symbiosis or artificially mixed together. This phenomenon is sometimes referred to as Castellani's phenomenon.

During the years 1904 and 1905, while in the East and in London on leave, I became interested in the study of baker's yeast and in fermentation caused by yeast. I made the following observations, mentioned at meetings of the Ceylon Branch of the British Medical Association.

1. Baker's yeast, both in the East and in London, is not a pure culture of a single species of yeast. It is generally composed of two or more species of yeast plus one or two or several species of bacteria. The bacteria isolated from samples of yeast in the East and in London do not produce gas-fermentation in sugars—they produce simple acidity.

2. Comparing the fermentation reactions of baker's yeast used in toto as obtained from the bakers, sowing the particles of the sample in the various sugars, with the fermentation reactions of the isolated pure cultures of the different species of saccharomyces and bacteria composing the sample, I found that the yeast in toto often ferments a larger number of carbohydrates than any one of the saccharomyces and bacteria that compose the sample.

3. By mixing artificially all the organisms isolated from the sample of yeast, in certain cases the same fermentation reactions are obtained as with the yeast in toto. There are apparent exceptions, as shown by the following example. From Orpington, a place in the country near London, I obtained in 1919 a sample of baker's yeast which gas-fermented a large number of carbon compounds, including starch. I investigated the sample mycologically by plating on maltose and dextrose agar, etc., and I isolated and grew in pure cultures all the organisms, saccharomyces and bacteria apparently present in that sample. By mixing all the pure cultures, I obtained the same fermentation reactions as with the sample of yeast in toto, except that the mixture did not ferment starch. The experiment was repeated many times with the same result, until finally I had recourse to the following procedure: the surface of the dextrose agar plates on which a minute particle of the yeast-cake had been plated out was washed with a little peptone water and the washing inoculated into starch. Fermentation took place, the inference being that probably some minute colonies on the verge of invisibility had escaped my notice when I investigated the plates and picked out all the colonies present; the thought came to me also that there might be an ultramicroscopic germ present.

4. By mixing artificially a nonmaltose fermenting saccharomyces (isolated from a sample of baker's yeast) with a bacillus isolated from the same sample, producing acidity in maltose and several other sugars, but never gas in any substance, I obtained fermentation of maltose with the production of gas.

5. In Ceylon by mixing two saccharomyces isolated locally, neither of which alone produced gas or acidity in maltose, gas fermentation of that sugar was obtained.

In recent years I have studied the same and similar phenomena, using mostly pathogenic bacteria: bacilli and cocci, also a pathogenic yeast.

"Symbiotic Fermentation Phenomenon."—As I have already stated, the phenomenon I have described may be defined as follows: two organisms, neither of which alone produces gas fermentation, may do so when mixed together; for instance, *Cryptococcus graciloides* alone will not produce gas in dextrose; *B. vermiculoides* alone will not produce gas in that sugar; when the two organisms live in symbiosis or are artificially mixed they will produce gas. This phenomenon is evident when certain bacteria are used; for instance, *B. typhosus* alone does not produce any gas in maltose (acid only); *B. morgani* alone does not produce any gas in that sugar (neither acidity nor gas): the mixture *B. typhosus* + *B. morgani* produces gas.

Use of the Symbiotic Fermentation Phenomenon in the Differentiation of Certain Micro-Organisms.—The symbiotic fermentation phenomenon may be used to differentiate certain micro-organisms; for instance, organisms of the dysentary group. The two principal types of dysentery organisms, Shiga and Flexner *sensu lato*, may be differentiated by the use of their symbiotic fermentation with *Bacillus morgani*. Shiga + Morgan will not produce gas in mannitol; Flexner + Morgan will produce gas. It may be said generally that the results of the symbiotic fermentation largely correspond to the results obtainable with simple acid fermentation, viz., there will be gas in those carbohydrates in which the organism to be identified will produce, alone, simple acidity; the presence of gas, however, is much more striking than simple acid fermentation; it may, moreover, in certain cases become rapidly evident, while the simple production of acid may be slow. For instance, there has been some difference of opinion on the point as to whether *Bacillus dysenteriae* Shiga-Kruse produces acidity in maltose. In old textbooks on bacteriology the organism is described as producing slight acidity in maltose; in most of the modern books it is described as not producing acidity. The symbiosis Shiga + Morgan produces gas in maltose, and this favors the possibility of Shiga producing a certain amount, however small, of acidity in that sugar.

Differentiation of Bacillus dysenteriae Shiga-Kruse, Bacillus dysenteriae Flexner, Bacillus dysenteriae Y (Hiss-Russell), by Means of Symbiotic Fermentation in Mannitol and Maltose.—The symbiosis Shiga + Morgan produces gas in maltose, not in mannitol; the symbiosis Flexner + Morgan produces gas in maltose and mannitol; the symbiosis Y + Morgan produces gas in mannitol, not in maltose.

Identification of Certain Carbon Compounds by Means of the Symbiotic Fermentation Phenomenon.—The phenomenon is useful in the identification and differentiation of certain carbon compounds. For instance, a Fehling-reducing substance which is gas-fermented by *Bacillus typhosus* + *Bacillus morgani,* and not by *Bacillus kandiensis* + *Bacillus morgani,* is probably maltose. The explanation is as follows: The symbiosis *Bacillus typhosus* + *Bacillus morgani* and the symbiosis *Bacillus kandiensis* + *Bacillus morgani* produce gas in the same Fehling-reducing sugars except maltose, in which *Bacillus typhosus* + *Bacillus morgani* produces gas and *Bacillus kandiensis* + *Bacillus morgani* does not. If a Fehling-reducing sugar, therefore, is gas-fermented by *Bacillus typhosus* + *Bacillus morgani* and not by *Bacillus kandiensis* + *Bacillus morgani* the inference is that it is maltose. For the identification of maltose and other carbon compounds, however, the mycologic method I devised in Ceylon and later worked out in England with Dr. F. E. Taylor is much simpler, as will be shown later.

Importance of Symbiosis in the Etiology of Certain Diseases and in the Causation of Certain Symptoms.—There are certain diseases which are, in all probability, caused by the symbiosis of two vegetal organisms such as a fungus and a bacterium; for instance, *Trichomycosis nigra* and *Trichomycosis rubra.*[14]

MYCOLOGIC METHOD TO DETECT AND IDENTIFY VARIOUS CARBOHYDRATES

Some years ago while I was in Ceylon I devised a mycologic method for the detection and identification of various carbohydrates. This method I worked out later on in London in the Kings College Laboratories with Dr. F. E. Taylor. A joint paper was published in the *British Medical Journal.*[15] Recently, we have published again a fairly complete description of the method in *The Journal of the American Medical Association.*[16] We do not have specific germs for each substance, germs, for instance, that will ferment only maltose, only galactose or only inulin, but as a number of fungi and bacteria select certain carbohydrates to the exclusion of others I found it possible, by using certain species and comparing the results, to devise a general method. To understand the working of the method it is necessary to keep in mind the biochemical fermentative characters of certain fungi

14. Castellani, Aldo: Fungi and Fungous Diseases; Lecture III, Arch. Dermat. & Syph., to be published.

15. Castellani, Aldo, and Taylor, F. E.: Brit. M. J. **2**:855 (Dec. 29) 1917.

16. Castellani, Aldo, and Taylor, F. E.: Fallacies of "Yeast Method" of Detecting Glucose in Urine, Further Observations on Mycologic Method to Identify Various Sugars and Other Carbon Compounds, J. A. M. A. **86**:523 (Feb. 20) 1926.

and bacteria, which are collected in table 11. I will limit myself to a brief description of the identification and detection of maltose, galactose and inulin, and I will also give a list of the principal mycologic formulas we have devised and employed in the identification of various carbon compounds.

Identification of Maltose.—In order to determine whether a given substance is maltose or not, a sterile 1 per cent solution in sugar-free peptone water is made and distributed into two tubes, each containing a Durham's fermentation tube or similar appliance. One of the following procedures is then used:

A. A 1 per cent sterile solution in sugar-free peptone water is made of the substance to be tested for maltose. The solution is distributed into two tubes, which are labeled 1 and 2. Tube 1 is inoculated with *Monilia tropicalis* and tube 2 with *Monilia macedoniensis*, or other monilias of the same group (*M. macedoniensoides*). If, after four days' incubation at 37 C., tube 1 contains gas and tube 2 does not, it can be concluded that the substance is maltose. This is easily understood if one keeps in mind the biochemical reactions of the two monilias. *M. tropicalis* ferments, with the production of gas, the following substances: dextrose, levulose, galactose, maltose and saccharose. *M. macedoniensis* and all other monilias of the macedoniensis group (*M. macedoniensoides*, etc.) ferment, with the production of gas, the following carbon compounds: dextrose, levulose, galactose, saccharose and inulin.

The substance being investigated has been fermented with the production of gas by *M. tropicalis*. It can be, therefore, only dextrose, levulose, maltose, galactose or saccharose, but, if it had been dextrose, levulose, galactose or saccharose, it would have been fermented with production of gas also by *M. macedoniensis*. Therefore, it can only be maltose.

$$\left.\begin{array}{ll} \textit{Monilia tropicalis} \text{ Castellani} & + \\ \textit{Monilia macedoniensis} \text{ Castellani} & 0 \end{array}\right\} = \text{maltose}$$

B. Tube 1 is inoculated with *Monilia pinoyi* and tube 2 with *Monilia krusei*. The two tubes are placed in an incubator at from 35 to 37 C. for three days. If, after that time, tube 1 (*Monilia pinoyi*) contains gas and tube 2 (*Monilia krusei*) does not, it can be concluded that the substance is maltose. This is easily understood if one keeps in mind the fermentative reactions of the two monilias. *Monilia pinoyi* ferments with the production of gas only the following carbon compounds: dextrose, levulose and maltose. *Monilia krusei* ferments, with the production of gas, only dextrose and levulose.

The substance under investigation has been fermented, with the production of gas, by *Monilia pinoyi;* it can be only dextrose, levulose or maltose, but if it had been either dextrose or levulose, it would have been fermented also by *Monilia krusei;* therefore, it can be only maltose.

$$\left.\begin{array}{ll} \textit{Monilia pinoyi} & + \\ \textit{Monilia krusei} & 0 \end{array}\right\} = \text{maltose}$$

C. Tube 1 is inoculated with *M. metalondinensis* and tube 2 with *M. macedoniensis* or *M. macedoniensoides*. The two tubes are incubated at 37 C. for four days. If, after that time tube 1 contains gas and tube 2 does not, the substance is maltose. This is explained by the fact that *M. metalondinensis* ferments with the production of gas only dextrose, levulose, maltose and galactose; and *M. mace-*

doniensis only the following carbon compounds: dextrose, levulose, galactose, saccharose and inulin.

The substance now being investigated has been fermented by *M. metalondinensis*. It can be, therefore, only dextrose, levulose, maltose or galactose; but, if it were dextrose, levulose or galactose, it would have been fermented also by *M. macedoniensis*. There is only one possibility left—it must be maltose.

Monilia metalondinensis Castellani +
Monilia macedoniensis Castellani 0
} = maltose

Identification of Maltose when Present with Other Common Fermentable Substances (Dextrose, Levulose, Galactose, Saccharose).—If a liquid is suspected of containing maltose mixed with dextrose, levulose, galactose and saccharose, it is fermented with *Monilia macedoniensis* or *M. macedoniensoides*. If after exhaustion with *M. macedoniensis*, the liquid is still fermented, with the production of gas, by *Monilia pinoyi*, *Monilia tropicalis* or *Monilia metalondinensis*, the conclusion is that the liquid also contains maltose. Strains of the various monilias should be used that have approximately the same fermentative power on the various carbohydrates they all ferment.

Identification of Galactose.—In determining whether or not a certain substance is galactose, the following procedures are used:

A. A 1 per cent sterile solution of the substance is made in sugar-free peptone water and distributed into two tubes, each containing a fermentation tube. Tube 1 is inoculated with *M. metalondinensis* and tube 2 with *M. pinoyi*. If, after four days' incubation at 37 C., tube 1 contains gas, and tube 2 does not, the substance must be galactose. The explanation lies in the fact that *M. metalondinensis* ferments only the following carbon compounds: dextrose, levulose, maltose and galactose; *M. pinoyi* ferments only the following substances: dextrose, levulose and maltose.

The substance has been fermented by *M. metalondinensis*. It can be, therefore, only dextrose, levulose, maltose or galactose. It has not been fermented by *M. pinoyi*, so it cannot be dextrose, levulose or maltose. There is only one possibility left—it must be galactose.

Monilia metalondinensis +
Monilia pinoyi 0
} = galactose

B. The solution is distributed into three tubes, each containing a fermentation tube. Tube 1 is inoculated with *M. metalondinensis*, tube 2 with *M. krusei* and tube 3 with *M. macedoniensis*. The three tubes are placed in an incubator at 37 C. for four days. If, after that period of time, tubes 1 and 3 contain gas and tube 2 does not, the substance is galactose.

This is easily explained if one keeps in mind the biochemical reactions of the three fungi. *M. metalondinensis* produces gas only in dextrose, levulose, maltose and galactose; *M. macedoniensis* produces gas only in dextrose, levulose, galactose, saccharose and inulin; *M. krusei* produces gas only in dextrose and levulose.

The substance now under investigation has been fermented, with the production of gas, by *M. metalondinensis*. It can be, therefore, only dextrose, levulose, maltose or galactose. It cannot be dextrose or levulose, because in such a case it

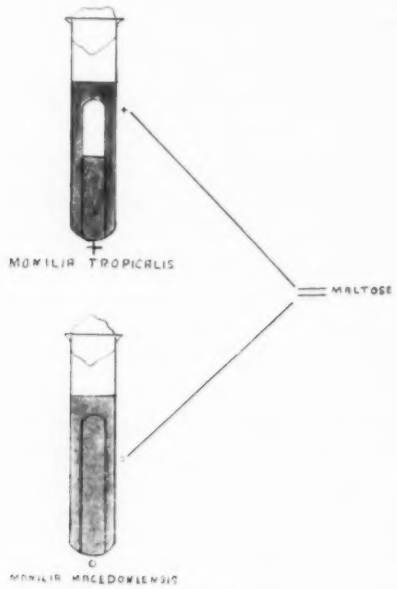

Fig. 44.—Identification of maltose.

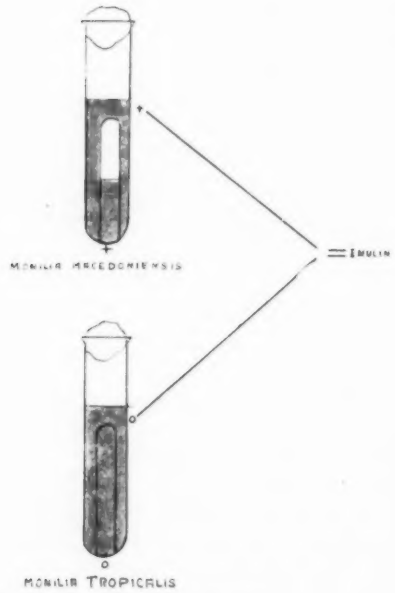

Fig. 45.—Identification of inulin.

would have been fermented by *M. krusei,* and it cannot be maltose, because if it were it would not have been fermented by *M. macedoniensis.* There is only one possibility left—it must be galactose.

Monilia metalondinensis	+	= galactose
Monilia krusei	0	
Monilia macedoniensis	+	

C. The solution is distributed into three tubes: Tube 1 is inoculated with *M. macedoniensis,* tube 2 with *M. krusei* and tube 3 with *B. paratyphosus* B. If, after four days in the incubator at 37 C., tube 1 shows presence of gas, tube 2 shows absence of gas and tube 3 shows presence of gas, it can be concluded that the substance under investigation is galactose. As the substance has been gas-fermented by *M. macedoniensis* it can be only dextrose, levulose, galactose, saccharose or inulin; but it has not been fermented by *Monilia krusei,* therefore it cannot be dextrose or levulose; it can only be galactose, saccharose or inulin, but had it been saccharose or inulin it would not have been fermented by *B. paratyphosus* B. There is only one possibility left—it must be galactose.

Monilia macedoniensis Castellani	+	= galactose
Monilia krusei Castellani	0	
Bacillus paratyphosus B	+	

D. The solution is distributed into two tubes; tube 1 is inoculated with *B. morgani* (strain S) and tube 2 with *Monilia krusei.* If, after three days in the incubator at 35 C., tube 1 contains gas and tube 2 does not, it can be concluded that the substance is galactose, provided the substance is known to be a carbohydrate.

This is easily understood by keeping in mind the fermentation reactions of the two germs. *Bacillus morgani* (strain S) ferments, with production of gas, glucose, levulose and galactose. The substance has been gas-fermented by *B. morgani;* if it is a carbohydrate it can only be dextrose, levulose or galactose. It has not been fermented by *Monilia krusei;* it cannot, therefore, be dextrose or levulose; it must be galactose.

B. morgani Castellani and Chalmers	+	= galactose
Monilia krusei Castellani	0	

Identification of Galactose When Present with Other Common Fermentable Substances (Dextrose, Levulose, Maltose, Saccharose).—To detect galactose in a liquid in which it is suspected to be present together with dextrose, levulose, maltose or saccharose, the liquid is inoculated with an organism which gas-ferments all the sugars I have mentioned except galactose; for instance, *Monilia bronchialis.* If, after exhaustion with *M. bronchialis* the liquid is still fermentable with *Monilia tropicalis,* the inference is that the liquid contained galactose, in addition to other fermentable substances.

USE OF MYCOLOGIC METHOD IN URINANALYSIS

1. The urine must be aseptic; if it cannot be collected aseptically, it should be sterilized as soon as possible after distribution in the tubes, by heating in Koch's steamer for thirty minutes. Once is sufficient for practical purposes, but in special cases, it may be heated on two or three

consecutive days. It should never be autoclaved, as this procedure may alter the character of the sugars and other carbohydrates present. Another procedure is to filter the sample of urine by means of a Chamberland or Berkefeld filter. I have recently found that boiling the urine for a few seconds does not apparently alter the chemical structure of the various sugars, and in practice this procedure is much simpler.

TABLE 13.—*Identification of Certain Fehling-Reducing Substances* *

Monilia balcanica Castellani	Monilia krusei Castellani	Monilia pinoyi Castellani	Monilia metalondinensis Castellani	Bacillus paratyphosis B Schottmüller	Bacillus coli Escherich	Reducing Substances
+	+	+	+	+	+	Dextrose
0	+	+	+	+	+	Levulose
0	0	+	+	+	+	Maltose
0	0	0	+	+	+	Galactose
0	0	0	0	+	+	Pentose
0	0	0	0	0	+	Lactose
0	0	0	0	0	0	Nonfermentable substances of the groups creatinine, hippuric acid, uric acid, etc.

* The reducing substance is made into a 1 per cent peptone water solution. Selected strains of the various organisms with permanent biochemical reactions and which produce a large amount of gas should be used.

TABLE 14.—*Key to Identification of Certain Fehling-Reducing Substances*

Inoculate tube 1 with Monilia balcanica Castellani
- + = Dextrose
- If negative inoculate tube 2 with M. krusei Castellani
  - + = Levulose
  - If negative inoculate tube 3 with M. pinoyi Castellani
    - + = Maltose
    - If negative inoculate tube 4 with M. metalondinensis Castellani
      - + = Galactose
      - If negative inoculate tube 5 with B. coli Escherich and tube 6 with B. paratyphosus B Schottmüller
        - B. coli 0, B. paratyphosus 0 → Nonfermentable reducing substances of the groups creatinine, uric acid, hippuric acid, etc.
        - B. coli +, B. paratyphosus B 0 → = Lactose
        - B. coli +, B. paratyphosus B + → = Pentose

2. It is of great advantage to add a third or the same amount of peptone water to the urine; otherwise, the organisms may, at times, grow scantily, and there may not be a distinct production of gas.

If a urine is Fehling-reducing, one can determine which of the six sugars found in pathologic urines (dextrose, levulose, maltose, galactose, lactose, pentose) is present, by the following procedure:

The urine is distributed into six tubes, each containing a Durham's fermentation tube, or saccharimeters may be used. They are labeled 1, 2, 3, 4, 5 and 6. Tube 1 should be inoculated with *Monilia balcanica* Castellani, or *M. parabalcanica* Castellani; tube 2 with *M. krusei* Castellani; tube 3 with *M. pinoyi* Castellani; tube 4 with *M. metalondinensis* Castellani; tube 5 with *B. coli-communior* Escherich, and tube 6 with *B. paratyphosus* B Schottmüller.

After the tubes are incubated at 35 C. for four days, the results may be read:

1. If in tubes 1, 2, 3, 4, 5 and 6 gas is present, the reducing substance is dextrose.

2. If tube 1 does not contain any gas, and in tubes 2, 3, 4, 5 and 6 gas is present, the reducing substance is levulose.

3. If tubes 1 and 2 do not contain gas, and in tubes 3, 4, 5 and 6 gas is present, the reducing substance is maltose.

4. If tubes 1, 2 and 3 do not contain gas, and in tubes 4, 5 and 6 gas is present, the reducing substance is galactose.

5. If tubes 1, 2, 3 and 4 do not contain gas, and in tubes 5 and 6 gas is present, the reducing substance is pentose.

6. If tubes 1, 2, 3, 4 and 6 do not contain gas, and in tube 5 gas is present, the reducing substance is lactose.

7. If none of the tubes contains gas, the reducing substance belongs to the nonfermentable group of reducing substances, including uric acid, creatinine, hippuric acid and others.

PRESENCE IN THE URINE OF MORE THAN ONE REDUCING SUBSTANCE

If the presence of more than one sugar and other carbohydrates is suspected, this may, to a certain extent, be ascertained and the various carbohydrates determined by the mycologic method. If it is assumed that the urine, after gas fermentation with *M. balcanica* Castellani, is still fermentable with production of gas by *M. krusei* Castellani, the conclusion is that in addition to dextrose the urine must have contained levulose; strains of *M. balcanica* Castellani, and *M. krusei* Castellani, having approximately the same fermentation power on dextrose should be used. If, then, after exhaustion, first with *M. balcanica* Castellani, and then with *M. krusei* Castellani, the urine is fermentable with production of gas by *B. morgani* Castellani and Chalmers the presumption is that it contained three sugars—dextrose, levulose and galactose. The explanation is found in the fact that *M. balcanica* Castellani ferments, with the production of gas, glucose only; *M. krusei* Castellani, dextrose and levulose; *B. morgani* Castellani and Chalmers, dextrose, levulose and galactose. If a Fehling reducing urine after exhaustion with *Monilia tropicalis* is capable of being fermented by *B. coli* and not by *B. paratyphosus* B, or is capable of being fermented by *Monilia pseudotropicalis*, it means that the urine in addition to dextrose, levulose, maltose or galactose, or a mixture of those sugars, contained also lactose.

Maltosuria.—Maltose, as is well known, reduces Fehling, and cases of maltosuria have naturally been mistaken for true glycosuria. For the detection of maltose, the urine is obtained aseptically by catheter, or if this is not feasible it is collected in a sterile receptacle and distributed at once in sterile tubes, each containing a small fermentation tube. The tubes are sterilized in Koch's stove for thirty minutes once or twice at an interval of six hours. This, in my experience, was generally sufficient, but at times it is advisable to repeat the

sterilization in Koch's stove on two or three consecutive days. The urine should never be autoclaved, as this procedure may alter the composition of the sugars and other carbohydrates present. The urine freshly passed may also be filtered through a Chamberland filter instead of being sterilized. The tubes of aseptic urine are inoculated with the monilias, as explained previously. If maltose is suspected to be present in conjunction with other fermentable substances, such as dextrose, levulose and galactose, the urine is exhausted with *Monilia macedoniensis*. If, after exhaustion with *Monilia macedoniensis*, the urine is still fermentable when inoculated with *Monilia tropicalis, Monila metalondinensis* or *Monilia pinoy*, it can be concluded that the urine contains maltose in addition to other fermentable substances.

REPORT OF ILLUSTRATIVE CASE

An Englishman, aged 47, a tea planter, had been a heavy beer drinker for many years. He was feeling quite well, except that his stoutness made it difficult for him to go for long walks, and this interfered with his duties. A substance that reduced Fehling's solution was found in his urine several times during the previous glycosuria which, it was thought, might develop into true diabetes. He came to me for consultation. The urine reduced Fehling's solution and was investigated for the presence of the following carbohydrates which reduce Fehling's solution: dextrose, levulose, galactose, maltose, lactose and pentose. The patient passed urine in my presence, into two sterile vessels; the urine of the second vessel was distributed immediately by means of a sterile pipet into five tubes of peptone water (two parts of urine to one of peptone water), each containing a fermentation tube. The tubes were labelled numbers 1 to 5. Number 1 was inoculated with *Monilia krusei* Castellani, number 2 with *Monilia pinoyi* Castellani, number 3 with *Monilia metalondinensis* Castellani, number 4 with *Bacillus coli* Escherich and number 5 with *Bacillus paratyphosus* B Schottmüller. The five tubes were placed in the incubator at 37 C. for four days. On examination the results were as follows:

Tube 1 *(Monilia krusei)*	0
Tube 2 *(Monilia pinoyi)*	+
Tube 3 *(Monilia metalondinensis)*	+
Tube 4 *(Bacillus coli)*	+
Tube 5 *(Bacillus paratyphosus* B)	+

This result according to my method means presence of maltose. The urine was gas-fermented by *Monilia pinoyi* and not by *Monilia krusei*. *Monilia pinoyi* produces gas only in three carbon compounds, viz., dextrose, levulose and maltose. the urine, therefore, must contain dextrose, levulose or maltose, or a mixture of these various sugars, but if the urine contained either dextrose or levulose it would have been gas-fermented also by *Monilia krusei*. The result obtained by my mycologic method was confirmed by the carrying out of purely chemical methods by a well known chemist.

Lactosuria.—Next to dextrose, lactose is the commonest sugar found in the urine. It is encountered frequently in the urine of nursing mothers. Lactose, as is well known, reduces Fehling's solution, and not infrequently lactosuria has been mistaken for true glycosuria. It is most important to distinguish between the two conditions, as lactosuria is not serious, while glycosuria, especially in nursing women, is of grave prognosis. The chemical detection and determination of lactose is difficult for an ordinary medical man. In most

textbooks it is stated that if a urine reduces Fehling's solution, and is not fermented by ordinary baker's yeast (so-called German yeast, or baker's yeast), the presumption is that the reducing substance is lactose. There are, however, two important sources of error.

First, a number of specimens of baker's yeast ferment lactose in addition to dextrose, maltose and other sugars; second, even if the baker's yeast should not ferment lactose, this is not the only substance that reduces Fehling's solution which may not be fermented by baker's yeast—pentose, for instance.

If a urine which reduces Fehling's solution is not fermented by ordinary yeast, it cannot, therefore, be concluded that the condition is lactosuria.

Detection and Determination of Lactose by the Mycologic Method.—This method can be carried out in various ways. A few mycologic formulas are cited as examples.

In a urine that reduces Fehling's solution, for practical purposes, the following formulas are correct:

I. *Bacillus coli* Escherich +
Bacillus paratyphosus B Schottmüller 0 } = lactose

Bacillus coli and *Bacillus paratyphosus* B, besides lactose, differ in their action on raffinose and salicine and glycerol, but raffinose and salicine are not carbon compounds found in the urine and at any rate they do not reduce Fehling's solution. The same formula applies to raffinose, salicine and glycerol, but there are substances which are of rare occurrence in the urine and, moreover, do not reduce Fehling's solution; therefore, this formula may be considered to be specific for practical purposes.

II. *Bacillus neapolitanus* Emmerich..... +
Bacillus pseudoasiaticus Castellani..... 0 } = lactose

The explanation lies in the fact that *Bacillus neapolitanus* and *Bacillus pseudoasiaticus* differ only in lactose as regards sugars and other carbohydrates found in pathologic urines.

III. *Monilia metalondinensis or Monilia tropicalis* Castellani 0 } = lactose
Bacillus coli Escherich..... + } (with great
Bacillus paratyphosus B Schottmüller..... ..... 0 } probability)

IV. *Monilia pseudotropicalis* Castellani +
Monilia macedoniensis Castellani 0 } = lactose

V. *Monilia pseudotropicalis* Castellani +
Monilia tropicalis Castellani 0 } = lactose

The technical details for carrying out the test are identical with those already mentioned in connection with the search for maltose, etc.

Pentosuria.—Pentosuria is not a rare condition. As is well known, the pentoses reduce Fehling's solution well and Nylander solution little; they give a positive reaction with Bial's and Read's modifications of the orcein test.

The mycologic method can be used for the detection of pentose in a urine that reduces Fehling's solution according to the following formulas:

Monilia tropicalis ..... 0 } = pentose
Bacillus coli Escherich..... + } (generally
Bacillus paratyphosus B..... + } arabinose)

Monilia metalondinensis..... 0 } = pentose
Bacillus coli Escherich..... + } (generally
Bacillus paratyphosus B..... + } arabinose)

REPORT OF ILLUSTRATIVE CASE

Some time ago I was consulted in London by a planter, aged 38, who had been sent home from a tropical country in the Far East, with his condition diagnosed as diabetes. His general condition, however, was fairly good, and he said that he had not lost much weight. According to his statement he was not thirsty or hungry, nor did he feel weak. He appeared somewhat nervous and irritable, however, and was worried at the idea of suffering from diabetes. The urine passed during twenty-four hours varied between 3 and 4 pints. It was slightly acid, with a density of 1.020. It persistently reduced Fehling's solution well, and apparently the diagnosis of diabetes had been based on this fact.

The urine was examined by the mycologic method for the presence of the following carbohydrates which reduce Fehling's solution: dextrose, levulose, galactose, maltose, pentose and lactose. The patient was made to pass urine into three sterile vessels; the urine of the second vessel was distributed immediately by means of a sterile pipet into six tubes of peptone water (about two parts of urine to one of peptone water), each containing a fermentation tube. The tubes were labelled numbers 1 to 5. Tube 1 was inoculated with *Monilia krusei* Castellani; tube 2 with *Monilia pinoyi* Castellani; tube 3 with *Monilia metalondinensis* Castellani; tube 4 wth *Bacillus coli* Escherich; tube 5 with *Bacillus paratyphosus* B Schottmüller. The five tubes were placed in the incubator at 37 C. and examined after forty-eight hours. The following were the results:

Tube 1 (*Monilia krusei* Castellani)	0
Tube 2 (*Monilia pinoyi* Castellani)	0
Tube 3 (*Monilia metalondinensis* Castellani)	0
Tube 4 (*Bacillus coli* Escherich)	+
Tube 5 (*Bacillus paratyphosus* B Schottmüller)	+

According to my method, these results in a urine that reduces Fehling's solution mean the presence of pentoses.

The result obtained by the mycologic test was confirmed by chemical investigation. The urine which reduced Fehling's solution well and Nylander solution little gave a positive reaction for pentoses with Bial's and Read's modifications of the orcein test. There can be little doubt, therefore, that the patient was suffering from "pentosuria" and not from diabetes. It is important in practice to try to differentiate in every case betwen glycosuria and pentosuria, as the first condition is often of grave prognosis, while the second points to a much more benign disturbance of metabolism and practically does not require any treatment.

Saccharosuria.—Spontaneous saccharosuria is an extremely rare condition, but artificial saccharosuria is not so rare among malingerers. During the Great War I encountered three cases in which saccharose had been added to the urine with the object of misleading the medical officer. The physician who uses as routine the usual Fehling method is not apt to diagnose such cases as glycosuria, as saccharose does not reduce Fehling's solution—at any rate not clearly; but the medical man who uses what is considered by most authorities the more exact method, that of yeast fermentation, will likely fall into error, because though in so many textbooks it is stated that dextrose is the only substance fermented by ordinary baker's yeast, I have found that a large number of specimens of baker's yeast will ferment saccharose and also levulose, maltose, etc., in addition to dextrose.

Mycologic Test to Determine Saccharose.—According to my method, various tests may be evolved to determine whether a certain carbohydrate of those found in pathologic urine is or is not saccharose. A few of these tests are presented here in the convenient shape of mycologic formulas:

I. *Monilia metalondinensis* Castellani.... 0
Monilia tropicalis Castellani.....+ } = saccharose

This is explained by the fact that *Monilia metalondinensis* and *Monilia tropicalis* are identical in all their biochemical reactions except on saccharose, which is fermented with production of gas by *Monilia tropicalis* and not fermented by *Monilia metalondinensis.*

II. *Monilia pinoyi* Castellani..... 0
Monilia rhoi Castellani.....+ } = saccharose

The explanation lies in the fact that *Monilia pinoyi* ferments, with production of gas, only dextrose, levulose and maltose, and *Monilia rhoi* only dextrose, levulose, maltose and saccharose. If a substance, therefore, is not fermented by *Monilia pinoyi* and is fermented, with production of gas, by *Monilia rhoi,* it must be saccharose.

To the foregoing purely mycologic formulas is added a chemicomycologic formula which may be found useful in practice:

Fehling 0
Monilia tropicalis Castellani...+ } = saccharose

This is explained by the fact that *Monilia tropicalis* ferments with production of gas, dextrose, levulose, maltose, galactose, saccharose and no other substance. If Fehling's solution gives a negative reaction, it cannot be any of the first four substances that is present, as they all reduce Fehling's solution; it must, therefore, be saccharose.

Detection of Saccharose when Mixed with Other Fermentable Substances.—The urine should be exhausted with *Bacillus coli* Escherich *sensu stricto* (does not ferment saccharose), and later, after filtration through a Chamberland filter, or prolonged centrifugalization, it should be inoculated with *Monilia tropicalis.* If the urine is still fermentable, the presumption is that in addition to other fermentable substances which can be found by the general method, the urine contains saccharose.

Inosituria.—Inosite, as is well known, does not reduce Fehling's solution; it is not really a sugar, and belongs to the aromatic series. It is not of rare occurence in the urine, and is, for instance, not infrequently found in diabetes insipidus, but the chemical methods of detecting it are far from easy or rapid. On the other hand, inosite, provided it is present in the urine in not too small a quantity, can be detected and determined with comparative ease by the mycologic method.

Two tubes of urine collected aseptically, or sterilized as already described, are mixed with one-third, or the same amount, of sugar-free peptone water and inoculated: number 1 with *Bacillus paratyphosus* B Schottmüller (M variety, which ferments inosite), number 2 with *Bacillus paratyphosus* A Schottmüller (does not ferment inosite). The two germs, as regards carbohydrates and other pathologic substances found in the urine, differ only in their action on ino-

site. Therefore, if, after forty-eight hours' incubation at 37 C., gas is present in tube 1, and absent in tube 2, one has to deal with inosite. This can be expressed conveniently by the following formula:

Bacillus paratyphosus B Schottmüller, var. M +
Bacillus paratyphosus A Schottmüller..... 0 } = inosite

If the result of the test is

Bacillus paratyphosus B Schottmüller, var. M..... 0
Bacillus paratyphosus A Schottmüller..... 0

it means that the substance is neither inosite nor any other substance fermentable by *Bacillus paratyphosus* B, var. M, or *Bacillus paratyphosus* A; hence, it cannot be dextrose, levulose, maltose, saccharose, etc.

If the result of the test is

Bacillus paratyphosus B Schottmüller, var. M.....+
Bacillus paratyphosus A Schottmüller.....+

the urine should be exhausted with *Bacillus paratyphosus* A, and then inoculated with *Bacillus paratyphosus* B, var. M, using strains having approximately the same fermentation power. If gas is produced, the inference is that the urine contained inosite in addition to other fermentable substances, which can be determined by using the general mycologic method.

SUMMARY OF THE GENERAL MYCOLOGIC METHOD

The entire subject can be condensed into the following formulas:

Mycologic Formulas

DEXTROSE

Monilia balcanica Castellani.....+ = dextrose

LEVULOSE

Monilia krusei Castellani.....+
Monilia balcanica Castellani..... 0 } = levulose

MALTOSE

Monilia tropicalis Castellani.....+
Monilia macedoniensis (Monilia macedoniensoides) Castellani..... 0 } = maltose

Monilia metalondinensis Castellani.....+
Monilia macedoniensis Castellani, or
Monilia macedoniensoides ..... 0 } = maltose

Monilia pinoyi Castellani .....+
Monilia krusei Castellani..... 0 } = maltose

Monilia pinoyi Castellani.....+
Monilia macedoniensis (Monilia macedoniensoides) Castellani..... 0 } = maltose

Monilia tropicalis Castellani.....+
Monilia rhoi Castellani..... 0 } = maltose

Monilia pinoyi Castellani.....+
Bacillus morgani Castellani and Chalmers .....0 } = maltose

Monilia metalondinensis Castellani.....+
Bacillus morgani Castellani and Chalmers .....0 } = maltose

GALACTOSE

Monilia metalondinensis Castellani.....+
Monilia pinoyi Castellani.....0 } = galactose

Monilia metalondinensis Castellani.....+
Monilia krusei Castellani.....0
Monilia macedoniensis Castellani.....+ } = galactose

Monilia tropicalis Castellani.....+
Monilia bronchialis Castellani.....0 } = galactose

Monilia macedoniensis Castellani.....+
Monilia krusei Castellani.....0
Bacillus paratyphosus B Schottmüller..+ } = galactose

Bacillus morgani Castellani and Chalmers .....+
Monilia krusei Castellani.....0 } = galactose

SACCHAROSE

Monilia tropicalis Castellani.....+
Monilia metalondinensis Castellani.....0 } = saccharose

Monilia tropicalis Castellani.....+
Bacillus coli-communis, sensu stricto (does not ferment saccharose) Escherich..0 } = saccharose

Monilia tropicalis Castellani.....+
Bacillus paratyphosus B Schottmüller 0 } = saccharose

Monilia macedoniensis Castellani.....+
Bacillus coli communis, sensu stricto Escherich 0
Bacillus coli communior.....+ } = saccharose

Monilia macedoniensis Castellani.....+
Bacillus paratyphosus B Schottmüller..0
Bacillus coli-communior.....+ } = saccharose

Bacillus coli-communis, sensu stricto Escherich 0
Bacillus neapolitanus Emmerich.....+ } = saccharose (with great probability)

Bacillus coli-communis, sensu stricto Escherich 0
Bacillus asiaticus Castellani.....+ } = saccharose (with great probability)

INULIN

Monilia macedoniensis or *Monilia macedoniensoides* Castellani.....+
Monilia tropicalis Castellani.....0 } = inulin

Monilia macedoniensis Castellani (*Monilia macedoniensoides*) ....+ *Monilia rhoi* Castellani.... 0	= inulin
Monilia macedoniensis (Monilia macedoniensoides) Castellani....+ *Bacillus coli-communior* Escherich; *Bacillus pseudocoli* Castellani; *Bacillus neapolitanus* Emmerich 0	= inulin
Monilia macedoniensis (Monilia macedoniensoides Castellani....+ *Bacillus asiaticus* Castellani.... 0	= inulin
Monilia macedoniensis (or *Monilia macedoniensoides* ....+ *Bacillus orleanensis* 0	= inulin

INOSITOL

Bacillus paratyphosus B, strain M Schottmüller ....+ *Bacillus paratyphosus* A Schottmüller.. 0	= inositol (with great probability)

GLYCEROL

Bacillus asiaticus Castellani....+ *Bacillus paratyphosus* B, var. M Schottmüller .... 0 *Bacillus colombensis,* strain L Castellani ....+	= glycerol (with great probability)

CHEMICOMYCOLOGIC FORMULAS

SACCHAROSE

Fehling's solution.... 0 *Monilia tropicalis* Castellani....+	= saccharose

LACTOSE

Fehling's solution ....+ *Monilia pseudotropicalis*....+ *Monilia macedoniensis* 0	= lactose
Fehling's solution ....+ *Bacillus paratyphosus* B Schottmüller... 0 *Bacillus coli-communis* (or *communior)* Escherich ....+	= lactose
Fehling's solution ....+ *Bacillus orleanensis* ....+ *Monilia macedoniensis* .... 0 *Bacillus coli* ....+	= lactose

PENTOSE

Fehling's solution....+ *Monilia tropicalis* Castellani.... 0 *Bacillus paratyphosus* B Schottt-müller ....+ *Bacillus coli-communis* Escherich....+	= pentose (generally arabinose)

As is the case with every other biologic method the results must be considered to be relatively and not absolutely certain.

ABRIDGED METHOD

The method I now use as a matter of routine is carried out using the following organisms:

1. *Monilia krusei* Castellani, 1909.
2. *Monilia tropicalis* Castellani, 1909.
3. *Monilia metalondinensis* Castellani.
4. *Monilia pinoyi* Castellani.
5. *Monilia pseudotropicalis* Castellani (strain IV).
6. *Monilia macedoniensis* Castellani, 1917 (var. *macedoniensoides*).
7. *Bacillus morgani* Castellani and Chalmers, 1918.
8. *Bacillus coli-communis* Escherich, 1886, *sensu stricto* (does not ferment saccharose).
9. *Bacillus coli-communior* Escherich-Durham, 1899 (var. *pseudocoscoroba*).
10. *Bacillus paratyphosus* B Schottmüller, 1902.

With the abridged method the following carbohydrates may be easily detected and identified: maltose, galactose, dextrose (or levulose) saccharose, lactose, pentose and inulin.

MALTOSE

Monilia tropicalis ..... + } = maltose
Monilia macedoniensis ..... 0 }

GALACTOSE

Monilia metalondinensis ..... + }
Monilia krusei ..... 0 } = galactose
Monilia macedoniensis ..... + }

Bacillus morgani ..... + } = galactose
Monilia krusei ..... 0 }

Monilia macedoniensis ..... + }
Monilia krusei ..... 0 } = galactose
Bacillus colombensis (L) ..... + }

Monilia macedoniensis ..... + }
Monilia krusei ..... 0 } = galactose
Bacillus paratyphosus B ..... + }

Monilia metalondinensis ..... + } = galactose
Monilia pinoyi ..... 0 }

SACCHAROSE

Monilia tropicalis ..... + } = saccharose
Monilia metalondinensis ..... 0 }

Monilia tropicalis ..... + } = saccharose
Bacillus paratyphosus B ..... 0 }

Monilia tropicalis ..... + }
Bacillus coli-communis, sensu stricto } = saccharose
(does not ferment saccharose) ..... 0 }

INULIN

Monilia macedoniensis (var. *macedoniensoides*) ..... +	= inulin
Monilia tropicalis 0	

LACTOSE

Monilia pseudotropicalis ..... +	= lactose
Monilia tropicalis 0	

Monilia tropicalis 0	= lactose (with great probability)
Bacillus paratyphosus B Schottmüller 0	
Bacillus coli-communis or Bacillus coli-communior ..... +	

DEXTROSE AND LEVULOSE

Monilia krusei ..... +	= dextrose (or levulose)

CHEMICOMYCOLOGIC FORMULAS

SACCHAROSE

Fehling's solution 0	= saccharose
Monilia tropicalis ..... +	

LACTOSE

Fehling's solution ..... +	= lactose
Monilia pseudotropicalis ..... +	
Monilia macedoniensis 0	

Fehling's solution ..... +	= lactose
Bacillus paratyphosus B 0	
Bacillus coli-communis ..... +	

Fehling's solution ..... +	= lactose
Bacillus paratyphosus B 0	
Bacillus coli-communior ..... +	

PENTOSE

Fehling's solution ..... +	= pentose (generally arabinose)
Monilia tropicalis 0	
Bacillus paratyphosus B ..... +	
Bacillus coli-communis or *Bacillus coli-communior* ..... +	

Cultivation of the Monilias Kept as Stock Cultures.—The organisms should be grown and kept at 22 C. or at the temperature of the room. Certain monilias, if kept too long in the incubator at 37 C., may lose some of their fermentative characteristics. The best medium is slightly acid dextrose agar. They should be transplanted once a month, and from time to time one should make plates to be certain that the cultures are pure.

KERATODERMA BLENNORRHAGICUM

CRITICAL REVIEW WITH A REPORT OF A CASE

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NEW YORK

Keratoderma blennorrhagicum is a rare skin disease. Only seventy cases are on record, of which eleven have been described in the American literature. In the following article, we have endeavored to clarify the confusion that still exists regarding pathogenesis, nomenclature and relationship to other similar eruptions.

Buschke,[1] in his monograph published in 1899, divided all gonorrheal manifestations on the skin into four groups: (1) simple erythemas, (2) urticaria and erythema nodosum, (3) hemorrhagic and bullous exanthems and (4) hyperkeratoses. This classification, though slightly schematic, has been generally accepted. The most important and characteristic group is the one of gonorrheal hyperkeratoses. On the basis of carefully studied cases, Buschke concludes that the hyperkeratoses should be strictly separated from other gonorrheal manifestations and should be classified as a separate clinical and histologic entity. The reasons supporting his conclusions are as follows: First, there are no transitory clinical or histologic manifestations which form a connecting link between the erythematous or exanthematous group on one hand and the hyperkeratotic group on the other. Second, this skin eruption is so unusual and its peculiarities so evident that there is no resemblance between gonorrheal hyperkeratosis and similar conditions of the skin of different etiology.

At the time of Buschke's publication, only six cases of gonorrheal keratoderma had been recorded. The first case, published in 1893 by Vidal,[2] was a classic description of this condition. He described the minutest details of his case, giving a picture of an unusual disorder of the skin, generalized and symmetrical, accompanied by a polyarthritis of a blennorrhagic origin. The small lesions, distributed all over the body, presented semitranslucent, dry papules and resembled droplets of

1. Buschke: Exantheme bei Gonorrhoe, Arch. f. Dermat. u. Syph. **48**:181, 1899.

2. Vidal: Eruption generalisée et symetrique de croutes cornées avec chute des ongles d'origine blennorrhagique, coincidant avec une polyarthritic de même nature. Recidive a la suite d'une guerison de la premiere maladie, Ann. de dermat. et syph. **4**:3, 1893.

wax. The larger lesions, particularly abundant over the upper and lower extremities, were peculiar. They formed stratified, dry, hard, cornified crusts resembling cutaneous horns, surrounded by an erythematous border, which could be lifted up by forceps, leaving a dry or slightly moist red area, verrucous at the base. The rupial character of the lesions prompted Vidal to consider them of syphilitic origin. On this assumption he instituted antisyphilitic treatment, which however, did not have any effect. A recurrence of the eruption two and a half years later, associated with the same polyarthritis and urethritis, led him to consider the entire syndrome as a separate clinical entity. Later reports of similar cases were published by other French authors, though the eruption in their cases was more localized. Vidal confined himself to a detailed clinical report without advancing any explanation of how the gonococcus might be responsible for this eruption. The theoretical speculation began with Chauffard [3] in 1897, who was also the first to perform a biopsy of this keratosis.

Baerman [4] was of the same opinion as Buschke. He thought that the etiologic connection was established beyond all doubt, because so far other hyperkeratotic skin manifestations had not the remotest resemblance to the peculiar eruption caused by the gonococcus. This conception is still prevalent and accepted by most authors, notwithstanding the fact that Buschke himself, in his subsequent monograph (1912), was forced to change the conclusions advanced in his earlier study. He discarded the strict separation of gonorrheal hyperkeratosis from other gonorrheal eruptions, because further careful observations revealed a hyperemic border surrounding the typical crusts and vesicular and pustular lesions here and there, preceding the evident keratotic lesions. Furthermore, the histologic picture studied by Chauffard and Sobotka [5] and recently by Keim [6] revealed an inflammatory process in the epidermis and papillary layer with the formation of vesicles filled with exudate. Those peculiar crusts presented stratified layers of a parakeratotic epidermis with the inclosure of numerous leukocytes, alternating with layers of dried exudate. On the basis of these histologic reports, Baerman doubts whether this keratosis should be conceived of as a true keratoderma. He thinks that the changes in the epithelium and papillary layer

3. Chauffard: Infection blennorrhagique avec Productions cornées de la peau, Bull. et mém. Soc. méd. d. hôp. de Paris **14**:93, 1897.

4. Baerman: Ueber hyperkeratotische Exantheme bei schweren gonorrhoischen Infectionen, Arch. f. Dermat. u. Syph. **69**:363, 1904.

5. Sobotka: Pustulos-hyperkeratotisches Exantheme bei gonorrhoischen Allgemeinerkrankung, Dermat. Wchnschr. **56**:181, 1913.

6. Keim: The Histogenesis of Keratoderma blennorrhagicum, Arch. Dermat. u. Syph. **9**:435 (April) 1924.

are of such a divergent nature that there is no resemblance between the histologic changes found in gonorrheal keratoderma and pictures found in other keratoses. With the aid of these clinical and histologic observations, it becomes evident that gonorrheal keratoderma belongs to the group of inflammatory keratoses.

Baerman also summarizes the clinical picture and differentiates two types of eruptions: the more frequent localized and the disseminated variety. In practically all cases, except that reported by Robert,[7] there is an involvement of the soles. French authors like to compare those localized keratotic plaques on the feet with a geographic relief map. This particular predilection to the soles was thought to be specific for blennorrhagic keratoderma. This conception can be easily disregarded when one considers the immobilization of the lower extremities on account of the polyarthritis. All of the cases observed have occurred in the male sex, with three exceptions: (1) Lee's [8] case of a woman, 63 years old; (2) Isaac's [9] case of a woman, 28 years old; (3) Robert's case of a girl, 4 years old. According to Bogrow,[10] polyarthritis was present in all of the cases reported, except in two of Robert's and in one of Roth's.[11] Arthritic complications did not occur in Robert's second case. His first patient, a girl aged 4, had a gonorrheal keratitis and conjunctivitis, and Roth's patient had a tendovaginitis. Bogrow found other gonorrheal manifestations in the 47 cases, such as tendovaginitis and bursitis, 5 cases; conjunctivitis, 9; iridocyclitis, 4; aortitis, 3; endocarditis, 3, and pericarditis, 2. Keim published a complete list of 59 cases reported until 1923. Further contributions to this interesting subject have been made by Mumford,[12] Oelze,[13] Du Bois,[14] Bussallac

7. Robert: Contribution a l'etude des troubles trophiques cutanée dans la blennorrhagie, These de Paris, April 28, 1897, Ann. de dermat. et syph. **8**:1053, 1897.

8. Lee: Keratodermia Blennorrhagica, Edinburgh M. J. **28**:99 (April) 1922.

9. Isaac: A Case of Keratodermia Blennorrhagica in a Woman, Brit. J. Dermat. **32**:195, 1920.

10. Bogrow: Ueber gonorrhoischen Keratosen, Arch. f. Dermat. u. Syph. **143**:44, 1923.

11. Roth: Zur Kasuistik des hyperkeratotischen gonorrhoischen Exantheme, München med. Wchnschr. **52**:104, 1905.

12. Mumford: A Mild Case of Keratodermia Blennorrhagicum, Brit. J. Dermat. **35**:462, 1923.

13. Oelze: Ueber Keratosis gonorrhoica, Arch. f. Dermat. u. Syph. **144**:1, 1923.

14. Du Bois: Keratoses blennorrhagiques on dermatites gonococciques, Acta derm.-venereol **5**:1, 1924.

Louigi,[15] Rille,[16] Sainz de Aja,[17] Willmott,[18] Garcia Faure,[19] Olivet,[20] Scholtz [21] and Bulkin.[22]

CASE REPORT

History.—D. S., a man, aged 32, chief adjuster, was born in Austria and lived in the United States since early childhood. He had been married for thirteen years, but did not have any children. He claimed that his wife had never had any miscarriages.

His father, aged 73, was living and well; his mother died at the age of 53 of diabetes mellitus. One brother died at the age of 42 of heart disease; another brother and five sisters were alive and well. There was no evidence of tuberculosis or cancer in the family. As a child, the patient had measles and at the age of 8, scarlet fever. Other details of his family history were irrelevant. About thirteen years ago, he had contracted acute gonorrhea. He immediately consulted his family physician and remained under his care for a period of four months. While he was being treated, a complicating epididymitis developed, but the joints were not affected. The treatment was intensive, consisting of irrigations, prostatic massages, sounds and internal medication, but he followed his occupation during this period. He was discharged apparently well and had no further complaint until the end of December, 1926, when he noticed a purulent discharge from the urethra. He then consulted a genito-urinary specialist, who instituted treatment for gonorrhea. He was well under treatment, when, at the end of January, 1927, he began to have pain around the joint of the ankle of the left foot. As time went on, his left ankle swelled, and the pain increased. He was soon unable to walk, and on the advice of his physician, he was admitted to the Hospital for Joint Diseases on February 14, where he remained until March 9. At the hospital, the routine treatment for urethritis and prostatitis was continued. He also received large doses of salicylates, combined with sodium bicarbonate, external applications of oil of wintergreen, electric baking, etc., which failed to improve his condition. Other joints became involved, and soon the inflammatory process extended to the right ankle joint, both knee joints and wrists and the joints of the little fingers. Two weeks later, at the end of February, the patient for the first time noticed lesions at the site of the swollen joints. They seemed to appear as small yellowish "pimples" which felt hard but did not cause any itching or pain. He said that a new crop of lesions always appeared a few days after a new joint became involved. The individual lesions grew larger, forming heaped-up, shell-like masses. At the beginning of March, his upper and lower

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15. Bussallac Louigi: Sapra un caso d'ipercheratosi blenorrhagica, Pathologica **16**:365, 1924.

16. Rille: Keratosis gonorrhoica, Deutsche med. Wchnschr. **50**:1568, 1924.

17. Sainz de Aja: Keratoderma blennorrhagicum, Actas dermosifilagr. **17**:103, 1925. (Spanish)

18. Willmott: "Keratodermie blennorrhagique" (Vidal), Arch. Dermat. et Syph. **13**:17, 1926.

19. Garcia Faure: Keratodermia blennorrhagica, Pseusa méd. Argentina **20**:739, 1925. (Spanish)

20. Olivet: Hyperkeratose und Thrombophlebitis bei Gonorrhoe, Med. Klin. **24**:916, 1926.

21. Scholtz: A Syndrome of Blennorrhagic Keratoderma, Arch. Dermat. & Syph. **15**:165, 1927.

22. Bulkin: Keratosis blennorrhagic a. Ruskij Westnik Dermat. **5**:2, 1927.

extremities were covered with similar lesions. At the same time, his scalp and chest became covered with greasy crusts which he could easily remove with his finger-nails. His penis became encased in a shell-like cast, which soon came off. The outer side of his left foot changed into one large horny "mass" which involved the little toe of the left foot. Heavy crusts formed under the nails of the fourth and fifth toes of the left foot, which were slightly painful. After the swelling of the joints had partly subsided, the patient left the hospital of his own accord. He was removed to his own home, where he continued to be under the care of his family physician, Dr. R. Goldberg. The eruption became worse and more generalized. We saw the patient in consultation after he had been confined to bed for a period of twelve weeks.

Examination.—The patient was well built. He lay in a recumbent and rigid position; he was pale and practically all the joints were involved and painful. Mentally, he was alert, answering questions intelligently, but he was extremely irritable and stubborn and was willing to co-operate only to a certain degree.

On the whole, the skin was pale. Over the swollen joints, however, it was glistening and of a yellowish hue. The eruption was fairly symmetrical and

Fig. 1.—Distribution of lesions on lower extremities. There are characteristic keratotic plaques of both feet.

generalized. The lesions appeared on practically every part of the body. There was, however, a marked predilection for the regions of the joints of the upper and lower extremities. The largest number of lesions was to be seen over the right shoulder joint. The largest individual formations of scales were over the right knee joint. Two types of lesions were distinguishable: large, irregular erythematous patches covered with a yellowish, heaped-up, but easily removable, crust. This type of lesion was prevalent in the scalp, neck, genitocrural and circumanal regions. The second type was a small pustule of a yellowish, waxy appearance, dry and firm, which soon grew peripherally, forming concentric scales, some of which resembled cutaneous horns, while others looked like oyster shells. The whole picture resembled closely a bird's-eye view of a mountain range.

Only the frontal part of the scalp was involved. The lesions could be followed across the hair line, especially on the left side of the forehead. The thick hair was full of greasy particles which were easily removed from the confluent areas covered with heavy yellowish crusts. Similar lesions were found on the upper and lower lip and on the anterior aspect of the neck. Here the lesions were moist, the crusts more heaped-up and the border of the whole area sharply marginated. The condition of the scalp, face and neck resembled strongly a

seborrhic dermatitis. The eruption on the chest consisted of fairly well circumscribed erythematous, oval or round lesions, 2 by 4 cm. in diameter; the border was made up of a thin crust. The involvement of the skin below the umbilicus was similar to that of the scalp and neck. The area involved was that over the genitocrural region, the penis, scrotum, circumanal region, buttocks and lumbar region; it showed the same heavy seborrhic crust, which at times left denuded, slightly moist areas on the surface.

We found the most characteristic group of lesions over the left knee. There were about fifteen lesions, either isolated or in pairs. Above the knee cap, they were coalescent, forming a large plateau. The more recent lesions presented a pea-sized papule, with the characteristic yellowish-gray scale. The scale was adherent with a slight depression in the center. On removal of the scale, a red,

Fig. 2.—Right knee, showing numerous lesions with cornified crusts.

slightly moist surface appeared which did not bleed. The largest efflorescence of the plateau above the kneecap consisted of a concentric heaped-up scale, about 1.5 cm. in diameter at the base and elevated about 0.5 cm. with a marked bluish-red hyperemic zone on the outer border. Following this hyperemic zone, a whitish ring, slightly raised above the surface, was noticed. The top of the lesion was not covered by yellowish-gray scales as in the more recent lesions, but was formed by cornified epidermis with a marked crateriform depression. The whole formation could be best studied on the dorsum of the left foot, where we saw three isolated lesions in different stages of development. The smallest lesion, more distally located above the head of the fifth metatarsal bone, presented a papule the size of a flax seed which was semitransparent and did not have a hyperemic zone; it resembled a droplet of yellow wax. The other lesions presented the characteristic stratified crusts.

The outer border of the left foot, the dorsa of the third, fourth and fifth toes and the plantar surfaces were markedly thickened and covered with tenacious, firmly adherent, grayish-yellowish scales. Similar hyperkeratotic changes were to be found on the right foot. In the middle of the planta, there was a pointed, concentric scale above the surface of the skin 1 cm. in height which strongly resembled a cutaneous horn. The surface of both feet gave the impression of a relief map. In the whole thickened area, the most pronounced changes were to be found on the little toe of the left foot. The entire toe seemed to be encased in a cornified mass; the surface was uneven and broken; it was verrucous at the base and covered with a more brittle crust around the nail. The nail itself

Fig. 3.—Right shoulder, showing largest number of small keratotic lesions.

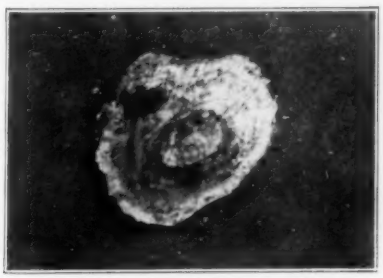

Fig. 4.—Typical rupial lesion.

was jagged and covered at the free border with a firmly adherent yellowish crust. On removal of these crusts—an entirely painless manipulation—a dry denuded surface was exposed.

Since a biopsy was refused, a scale, as shown in figure 4, was removed and submitted for pathologic study. A cross-section of the rupial scale showed a large number of layers of flattened epithelial cells irregularly arranged. Some of these showed large pale-staining nuclei; others contained irregularly shaped granules of chromatin that stained deeply with hematoxylin. In some areas, there were large homogeneous masses with here and there free epithelial plates, some still containing pale blue nuclei. At one end, there were individual keratinized whorls surrounded by densely stained material (Dr. Samuel A. Goldberg).

Results of the examination of the genito-urinary tract made by Dr. Joseph A. Lazarus were as follows: A number of pus cells were found in the urine. The urethra admitted a twenty-four (24) bougie. The prostrate was enlarged, and the secretion contained thick pus but no gonococci. Roentgen-ray examination of the genito-urinary tract revealed that the right kidney was slightly enlarged and greatly ptosed, its lower border reaching to about 0.5 cm. above the crest of the ilium. The left kidney was about twice the normal size. The spleen was also enlarged. Evidence of calculus was not found in any part of the genito-urinary tract.

The patient was lying in a recumbent position with both legs rotated outwardly. The knees and hips were flexed. The knee joints were moderately enlarged.

Fig. 5.—Photomicrograph showing nucleated and non-nucleated layers of flattened epithelial cells with two keratized whorles at one end.

There was some local heat but no signs of hyperemia. Fluctuation could not be elicited. Crepitation on active movement was absent. Active and passive movements were limited. The ankle joints were enlarged but not painful. The joints of both wrists were much swollen, not tender to pressure, but painful when active or passive movements were made. The elbow joints were comparatively free; all finger joints were involved. Active and passive movements in the shoulder joints were limited and painful.

Roentgenography of the right ankle joint showed a dense synovitis and a marked peri-articular swelling without evidence of active destruction of the joint surface of the bones. The diagnosis was infectious arthritis.

Examination of the heart revealed normal cardiac dulness and normal sounds; a murmur was not heard. The blood pressure was 118 systolic and 64 diastolic. There were normal breath sounds and no areas of dulness.

The pupils reacted to light and accommodation. There was slight exaggeration of the patellar reflexes. Hearing and eyesight were good.

Laboratory Examination.—The urine was examined on several occasions. There was a faint trace of albumin, acid reaction, a specific gravity of 1.026 and

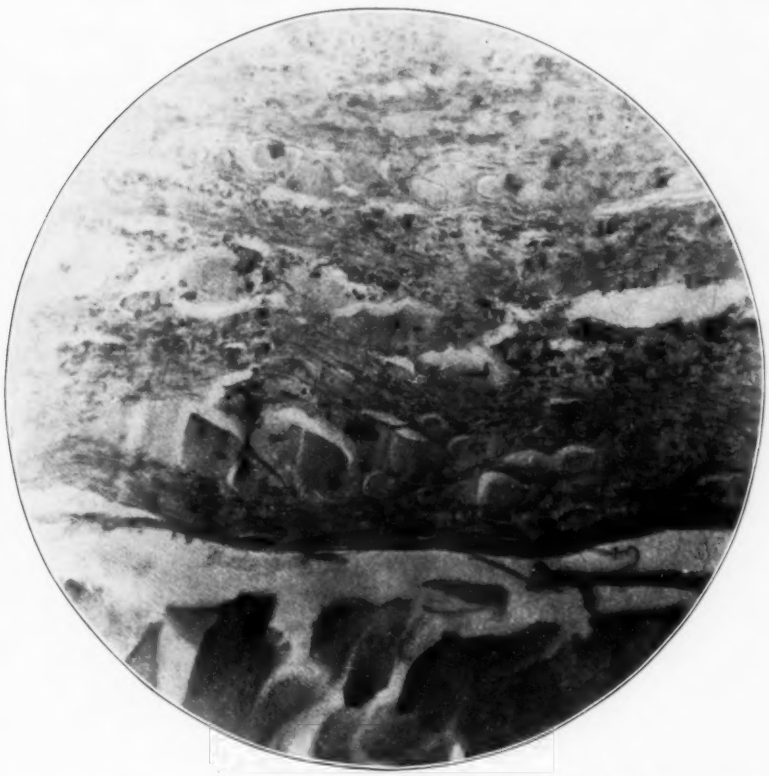

Fig. 6.—High-power magnification showing homogeneous keratized masses: layers of flattened epithelial cells and interspersed areas of large epithelial plates with and without nuclei.

many pus cells, but no casts. The Wassermann reaction and the complement-fixation test for gonorrhea were negative. The blood examination showed: hemoglobin, 60 per cent; red blood corpuscles, 4,448,000; white blood corpuscles, 14,900; neutrophils, 71 per cent; small lymphocytes, 19 per cent; large lymphocytes, 10 per cent. The Widal reaction was negative, and the blood culture was sterile.

Course and Treatment.—The treatment instituted at the beginning was directed particularly to the genito-urinary tract and the complicating polyarthritis. We have resorted to local treatment consisting of hot fomentations, shaking mixtures

and ointments, which were of little benefit. With the improvement in the joints, the involution of the cutaneous lesions began. Striking improvement was noted after a prostatic abscess broke through the rectum and drained profusely. Examination did not reveal gonococci. Vaccine therapy had been used at the hospital with little effect. The patient received injections of a mixed gonococcic vaccine, 1 cc. subcutaneously, at intervals of two or three days.

PATHOGENESIS

In order to explain the pathogenesis of this disease, a trophoneurotic and an infectious theory were advanced. The first was based on the symmetry of lesions, on the symmetrical disturbance of the affected joints and negative bacteriologic observations made on the blood and on the cutaneous lesions. The infectious theory has been advanced by those who have found the gonococcus either in the blood stream or in the skin. In spite of those observations, the question as to whether or not one is permitted to make the neisserian diplococcus directly responsible for the production of those unique and imposing cornified crusts still remains unanswered. Among the seventy cases of authentic keratoderma blennorrhagicum reported, we find that only Wadsack [23] succeeded in demonstrating gram-negative diplococci in the tissue in a true case of keratoderma blennorrhagicum. This isolated report prompted sincere observers to accept the conclusion that, as Buschke stated, this is a definite proof of true gonococcic invasion, at least in a number of cases

The modern conception of the development of this eruption is based on an analogy with other gonorrheal manifestations, such as gonorrheal arthritis. Three types of the condition can be distinguished.

Keratoderma Blennorrhagicum of Toxic Origin.—This type is characterized by the absorption of toxins from a focus (seminal vesicles), a short incubation period and a stormy course of the urethritis. Numerous joints and tendons are affected, but there is very little exudate. Gonococci are absent because of the strong auto-immunization. The complement-fixation test is strongly positive because of the presence of a large amount of antibodies. Therefore, the treatment with specific vaccine should be started in the so-called negative phase after the infection clears up. Garcia Faure [19] bases the explanation of the syndrome of gonorrheal keratoderma on this theory. He assumes that in gonorrhea with visceral and arthritic complications there is an abundant production of toxins. Those toxins produce a disturbance in the colloidal balance and a sensibilization of certain parts of the skin. When they are carried to these sensitized cells, a proliferation of pathogenic cells results, which finds its expression in the lesions of blennorrhagic keratoderma. After the disappearance of the clinical eruption, however, the hypersensitiveness of the cells still remains. Thus, the reappearance of new lesions with every new infection is easily understood.

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23. Wadsack: Ein gonorrhea Exanthem, Berl. Klin. Wchnschr. **43**:966, 1906.

Keratoderma Blennorrhagicum of Metastatic Origin.—The incubation period is longer than in the toxic form of the condition. The course is usually mild. As a rule, only one joint becomes affected. The joint contains pus, and gonococci are also occasionally found. The complement-fixation test is weakly positive. Treatment with large doses of vaccine is indicated and is successful. Bogrow [10] considers the metastatic origin as the most plausible. This explains the simultaneous occurrence of metastatic infections of other organs and the frequent coincidence of the eruption and other metastatic processes.

Keratoderma Blennorrhagicum of Septic Origin.—This type, which interests us mostly, is characterized by a long incubation period, a torpid course of the urethritis, involvement of numerous joints and the prolonged course of the polyarthritis. Bordet's and Gengou's phenomena are negative. Because the patient is not able to produce antibodies, the treatment with vaccines is without any scientific foundation. We consider our case as one of a low grade septicemia. The negative bacteriologic observations on the blood and rupial crust, and the negative complement-fixation test could be explained by the inability of the patient to mobilize his forces of defense, i. e., to produce antibodies. The chronicity of the gonorrheal infection and the long interval between the outbreak of the latter and the eruption on the skin support our conception.

On the basis of this classification, important observations which have caused considerable controversy could be explained. We can understand why in one case the complement-fixation test is positive and in the other negative; why vaccine treatment is indicated and helpful, or why, because of the absence of auto-immunization, it is of no avail.

RELATIONSHIP WITH ARTHROPATIC PSORIASIS

The question of blennorrhagic keratoderma versus arthropatic psoriasis has been thoroughly discussed in the literature. Adamson's [24] opinion is usually cited in support of the view that the conditions are identical. But long before him, Buschke [25] published a series of cases which, clinically and histologically, presented every detail of the syndrome of blennorrhagic keratoderma. There was the same hyperkeratosis on an inflammatory base and the striking similarity of the individual lesions, from the smallest keratotic papules to the largest cornified crusts, so typical of keratoderma blennorrhagicum. Polyarthritis and a subacute infection of the genito-urinary tract were likewise present. In other words, the triad-hyperkeratosis, polyarthritis and genito-urinary infection—which some authors consider a condition sine qua non for the

24. Adamson: Keratodermia Blennorrhagica: Is It a Form of Psoriasis? Brit. J. Dermat. **32**:183, 1920.

25. Buschke: Ueber universell symmetrische entzündliche Hyperkeratosen auf uro-septischer und arthritischer Basis, Arch. f. Dermat. u. Syph. **113**:223, 1912.

diagnosis of a true keratoderma blennorrhagicum, was demonstrated in those patients despite the absence of a gonorrheal infection. These cases could not be classified as cases of keratoderma blennorrhagicum. Consider Adamson's cases. In a true case of blennorrhagic keratosis, he observed lesions on the trunk and extremities that strikingly resembled psoriasis. Naturally the question arose whether this case should not have been classified as psoriasis arthropatica. In other cases studied by this author in which the diagnosis of psoriasis arthropatica was made, but in which the lesions on the palms and soles resembled those of a gonorrheal keratosis, the diagnosis was questionable. There are numerous reports in the literature in which the cases are unhesitatingly classified as gonorrheal keratoderma because the clinical picture corresponds so well with the original report of Vidal; and rightly so. Clinically, it is comparatively easy to recognize the characteristic syndrome. The real difficulty arises when an attempt is made to prove that this unique clinical picture is gonorrheal in spite of all negative bacteriologic observations and in spite of the resemblance to arthropatic psoriasis. Some authors, not being able to convince themselves concerning the gonorrheal etiology and seeing the similarity of the lesions to those classified as psoriasis arthropatica, went to the extreme of considering all cases of blennorrhagic keratoderma as a form of psoriasis arthropatica. This view is brought out in the recent study by Falk,[26] to which we cannot subscribe. Our contention is that the syndrome must be considered clinically and histologically as an entity of various etiology. In the majority of cases, it has been observed in association with gonorrhea. The same picture is found at times in patients suffering from other infections of the genito-urinary tract and also in those suffering from psoriasis. Whatever the etiology may be, the clinical syndrome is always the same, with the exception of minor differences such as those found in other diseases of the skin.

NOMENCLATURE

The nomenclature of this syndrome has undergone various changes in the hands of different authors. Numerous names have been substituted for the term "keratoderma," and the adjective "blennorrhagicum" has not been left untouched. The use of the synonyms "keratodermia," "keratosis," "hyperkeratosis," "dermatitis," "exanthema" and "hyperkeratotic exanthema" indicates the dissatisfaction with the term "keratoderma." In order not to commit themselves, some describe the syndrome as Vidal [2] did originally, "Eruption generalisée et symetrique de croutes cornées avec chute des ongles d'origine blennorrhagique, etc." "Blennorrhagicum" is found more appropriately expressed thus: "in a

26. Falk: Psoriasis arthropatica (einschliesslich der sog. hyperkeratotischen Exantheme bei gonorrhoischen Gelenkerkrankungen), Arch. f. Dermat. u. Syph. **129**:299, 1921.

blennorrhagic or gonorrheic patient" or "in gonorrhea." The German authors often use the expression "in gonorrhoischer Algemeinerkrankung."

Early observers have expressed doubt as to whether the condition is a true keratosis, and careful microscopic studies have helped to disperse the misleading conception of a true keratosis or hyperkeratosis. Baerman [4] proposed the name "dermatitis papillaris parakeratotica." This term expressed the true nature of the histologic observations but because it was clinically unsatisfactory, it was not accepted. Recently, Du Bois [14] came to the conclusion that this dermatologic entity was produced through capillary embolism of a bacteriologic nature which leads to peripheral distribution on the basis of an individual predisposition of the skin. He was also convinced that the disease does not contain any true keratotic elements and so he suggested the name "dermatitis gonorrhoica." These various terms fail to convey the true nature of this clinical entity. We, therefore, suggest that the name "dermatitis rupioides arthropatica" should be generally adopted. This term does away with the misleading name "keratoderma" and the questionable etiologic factor. Clinically and histologically, the condition is a dermatitis, beginning with a vesicle, changing into a pustule and finally developing into the characteristic rupial lesions. The arthropathy is nearly always present, and is of assistance in making correct diagnosis. We have discussed the fact that the syndrome cannot be considered rigidly as a gonorrheal manifestation as the term "blennorhagicum" would indicate. We are convinced that various etiologic factors are responsible for this clinical entity.

SUMMARY

1. Seventy cases of keratoderma blennorrhagicum are on record, our case being the twelfth published in the American literature on the subject.

2. Careful research has shown that keratoderma blennorrhagicum is not a true keratosis but an inflammatory process, which begins with a vesicle which dries up and forms a characteristic keratotic lesion.

3. A case in which there is a generalized eruption with a large number of rupial lesions is described in detail.

4. Gonorrhea is not alone responsible for the syndrome of keratoderma blennorrhagicum. Other infections of the genito-urinary tract and certain cases of psoriasis arthropatica simulate this picture clinically and histologically beyond recognition.

5. Unnecessary discussion could be avoided if the etiologic factor in the terminology and the misleading term, "keratoderma," could be discarded. We, therefore, suggest that a name which conveys the true clinical picture should be adopted, i. e., "dermatitis rupioides arthropatica."

Editorials

SUPPOSED DANGERS IN WET DRESSINGS OF ALUMINUM ACETATE

In the June, 1927, number of the *American Journal of Surgery* (p. 573), there is an article by Dr. Arthur E. Hertzler on the dangers in the use of aluminum acetate solution as a wet dressing, which must be a surprise to dermatologists. In this paper is recorded a case in which gangrene was produced by a wet dressing of a weak solution of this well known, usually bland dressing. It was a case of infection of the finger in which the infection extended only to the subcutaneous tissue. A solution of aluminum acetate 30 grains (1.95 Gm.) to a pint of water was applied. When the patient was seen a week later, the skin on the dorsal surface, particularly over the knuckles, was badly macerated, the distal portion of the index finger was black as far as the base of the second phalanx, and amputation of the finger was necessary. Microscopic examination showed endarteritis and complete thrombosis of the veins. Hertzler refers to the same condition illustrated in a monograph by Klapp and Beck, Leipzig, 1923, plate 6, and cautions against too long use of aluminum acetate solution. He concludes: "Generally speaking all would be accomplished in 24 hours that can be accomplished with this drug and when prescribed a limitation should be placed on its use. The same general rule is applicable to all wet dressings."

The only interpretation one can place on the last sentence is that any wet dressing exposes the patient to the dangers of necrosis if used more than twenty-four hours. If that is true, the danger may be not in the use of aluminum acetate solution, but in the use of any wet dressing.

The application which he used was "a solution of aluminum acetate, 30 grains to a pint of water." This leads to confusion as to whether there was any aluminum acetate in the solution. Aluminum acetate is insoluble in water. The official solution of aluminum acetate is not made by simply putting aluminum acetate salt in water, but by double decomposition in a mixture of aluminum sulphate, acetic acid, calcium carbonate and water.

The aluminum acetate wet dressing is probably one of the applications most frequently used by dermatologists. It has been a standby, as Burow's solution, beyond the memory of any dermatologists now living. It would be interesting to know whether any dermatologist has ever seen necrosis result from its use. *A priori*, it would not be expected; it is not analogous to weak solutions of phenol. A solution of one part

of the 8 per cent solution—the standard solution of the National Formulary—in sixteen parts water, that is, a true 0.5 per cent solution, which is the one ordinarily used by dermatologists, is almost as bland as physiologic sodium chloride solution. It may produce slight irritation in sensitive patients, but this irritation is so slight that it is hard to conceive that it may cause serious damage even to the superficial structures. When endarteritis and thrombosis of the deeper veins are produced, it is hard to understand how it could do it.

The incrimination of all wet dressings as dangerous in the statement that "the same general rule is applicable to any wet dressing" would seem to indicate that the author is not certain in his own mind that the aluminum acetate solution was the cause of the gangrene, and that he is inclined to regard all wet dressings as potential producers of this condition. In view of the fact that a wet dressing which in effect is hardly more bland than, say, physiologic sodium chloride solution, completely envelops the human body for months before it emerges into the open air, it would seem that this contention of the danger of all wet dressings must be a surprise to dermatologists and to others.

Another explanation for the gangrene in Hertzler's case is that the infection of the fingers might have produced gangrene. Why is not that the explanation of the endarteritis and thrombosis in his case?

To summarize, then, the points against the incrimination of aluminum acetate in this case are: first, if aluminum acetate salt itself was used in making the solution for the wet dressing there was no aluminum acetate solution, because the salt is practically insoluble in water; second, there are other more plausible factors to account for the gangrene.

The matter is not one of mere academic interest to dermatologists. Aluminum acetate solution is one of the most useful solutions. It is highly important that one should not interfere with its use by giving it a reputation of having serious dangerous possibilities when they do not exist.

We should be glad to hear the experience of any dermatologist pertinent to the question raised in this report.

W. A. P.

THE HISTOLOGIC DIAGNOSIS OF CUTANEOUS CANCER

In the issue of June 18, 1927, of the *Journal of the American Medical Association*, Dr. C. White and Dr. F. D. Weidman published an article entitled "Pseudo-Epitheliomatous Hyperplasia at the Margins of Cutaneous Ulcers with Special Reference to Histologic Diagnosis." It brings up a matter of great importance, namely, the unreliability of histologic diagnosis of epithelioma in ulcers of the skin. The gist of the article is as follows: Epithelial hyperplasia at the margin of non-

malignant cutaneous ulcers may mimic malignant changes so closely that the histologic diagnosis of epithelioma may be made where epithelioma actually does not exist. The authors' studies on this subject show that the final decision must be made on the basis of the clinical behavior of the lesion. They describe types of nonmalignant hyperplasia, from that of pronounced acanthosis to those showing all the characteristics of squamous cell cancer. The two histologic features that they record as of the most importance in the diagnosis of cancer are the embryonic character of the cells and their deep penetration of the corium. These two features when interpreted by a pathologist with experience in epidermal hyperplasia may justify the diagnosis of malignancy. The authors feel, however, that probably many times diagnoses are made of beginning carcinoma on the margins of ulcers when actually only exaggerated regenerative hyperplasia is present, and that, as a consequence, unnecessary radical treatments are carried out. This last expression of opinion must accord with the experience of all dermatologists. There is hardly any one of us who has not seen cases in which radical operation was contemplated by competent surgeons when the clinical diagnosis was clearly not carcinoma.

This statement of uncertainty in the diagnosis of epithelioma in ulcers of the skin by experts of such high skill in cutaneous pathology will undoubtedly strike a responsive cord in many dermatologists who have been impressed with the importance of the clinical diagnosis in epithelioma. There is a general attitude that the diagnosis of epithelioma must be confirmed by a histologic examination. But here comes a statement from the most expert pathologists in cutaneous diseases and in cutaneous pathology that the histologic diagnosis must be regarded as uncertain and that the reliable diagnosis is the clinical one. This accords with dermatologic experience. Few lesions are more characteristic in their macroscopic appearance and in their course than cutaneous epitheliomas. In calling attention to the preponderating importance of the clinical features in making a diagnosis and to the mistakes that are likely to occur in cutaneous ulcers by too great a reliance on a histologic picture that may mimic epithelioma, the authors have emphasized an important fact which is not generally recognized.

W. A. P.

Abstracts from Current Literature

RAYNAUD'S DISEASE WITH OCULAR COMPLICATIONS. S. J. APPELBAUM and M. L. LERNER, Am. J. Opth. **9**:569 (Aug.) 1926.

The author gives the report of a case in addition to the original paper. The cutaneous symptoms of the disease were preceded by migraine, visual disturbances and temporary loss of memory. There was bilateral papilledema and associated retinal hemorrhages and exudates.

TERTIARY SYPHILIS OF EYELID. E. S. CONNELL, Am. J. Ophth. **9**:608 (Aug.) 1926.

A case of tertiary nodular syphilis reported because of the infrequency of isolated lesions of this type.

ITCHING OF EYELIDS DUE TO ALLERGIC REACTION. F. W. MARLOW, Am. J. Ophth. **10**:52 (Jan.) 1927.

A case report is given in which itching of the eyelids followed the use of orris root powder on the scalp. No other cutaneous manifestations were recorded.

CONJUNCTIVITIS AND DERMATITIS DUE TO BUTYN. A. N. LEMOINE, Am. J. Ophth. **10**:125 (Feb.) 1927.

A case report is given in which conjunctivitis and dermatitis followed instillation of a 1 per cent butyn solution. A positive intradermal test reaction was obtained.

TEMPORARY ANISOCORIA IN A LEPROSY PATIENT. B. BHADURI, Am. J. Ophth. **10**: 194 (March) 1927.

Bhaduri reports a case of temporary anisocoria in which the supra orbital area of the affected eye showed cutaneous lesions and cutaneous anesthesia; the opposite side was hyperesthetic and the eye normal.

PEMPHIGUS OF BOTH EYES. MICHAEL GOLDENBURG, Am. J. Ophth. **10**:251 (April) 1927.

A woman, aged 56 (nationality not noted), had lesions confined to the ocular conjunctiva, cornea and gingival and buccal mucosa. She was presented before the Chicago Dermatological Society by Dr. Stillians, and the diagnosis confirmed. Some response followed the Davis treatment, though there were repeated relapses.

SARCOID OF THE EYE-LIDS. L. LEHRFELD, Am. J. Ophth. **10**:255 (April) 1927.

A case is reported in which the nodules on the eyelids were part of an extensive cutaneous eruption. Microscopic examinations for tubercle bacilli in sections of the tissue and in fluid from a hydroxthorax were negative.

RESULTS WITH INTRACISTERNAL INJECTIONS IN LUETIC OPTIC ATROPHY. S. R. GIFFORD and J. J. KEEGAN, Am. J. Ophth. **10**:323 (May) 1927.

A report is given concerning six patients with tabetic optic atrophy who were treated prior to 1923, and eight others treated since then. This method was employed following failure of intensive arsphenamine and mercury medication including spinal drainage and the Swift-Ellis method of treatment.

One tenth cubic centimeter of a 1:200 solution of mercuric chloride diluted with from 15 to 20 cc. of spinal fluid was injected once in six weeks, and from three to five doses were given. The day following each injection, neoarsphenamine was administered intravenously, and during the interval between injections inunction of mercury and of potassium iodide were employed. Marked meningeal irritation occurred in several cases, but no serious complications followed. The patients were hospitalized from twenty-four to forty-eight hours.

Improvement was noted in six cases; the condition remained stationary in five after treatment was instituted, and in two there was some progression of the disease process, though blindness did not occur. The authors conclude that cisternal injections have a distinct advantage over intraspinal treatment, and that intravenous treatment alone is inadequate in cases of progressive optic atrophy.

THRUSH OF THE CONJUNCTIVA. A. H. NORTON, Am. J. Ophth. **10**:357 (May) 1927.

A case report is given of a boy, aged 5, with thrush of the conjunctiva.

CONJUNCTIVITIS AND DERMATITIS DUE TO BUTYN. F. H. NEWTON, Am. J. Ophth. **10**:432 (June) 1927.

A report of a case is given.

CASE OF OCULAR LEPROSY. R. M. ROGERS, Am. J. Ophth. **10**:503 (July) 1927.

Ocular leprosy and its treatment is discussed. Observations on the condition occurring in a Russian after twenty-five years of residence in the United States are included in the discussion.

CHANCRE OF CONJUNCTIVA WITH PARENCHYMATOUS KERATITIS. S. J. MEYER, Am. J. Ophth. **10**:594, (Aug.) 1927.

The author reviews the literature on this subject and gives a report of two cases. In both cases spirochetes were found on dark-field examination. The keratitis was considered a result of direct spirochetal invasion of the cornea, though other investigators have attributed it to the toxemia resulting for the sudden destruction of spirochete with arsphenamine. Intensive treatment with neoarsphenamine, and mercury or bismuth, is advocated. Chancres of the eye are said to make up about 4 per cent of extragenital infections.

FOERSTER, Milwaukee.

BROMIDE INTOXICATION: FURTHER STUDIES. U. J. WILE, J. A. M. A. **89**:340 (July 30) 1927.

Wile briefly reviews his previous contributions on this subject and adds further clinical evidence (eight cases) showing the value of injections of sodium chloride. He has found a new test (described by Belote in another paper) of value in the detection of bromine in all body fluids. The initial dose of sodium chloride solution intravenously should not exceed from 100 to 150 cc. This may be increased to 400 cc. if well tolerated. Occasionally renal irritation occurs as a result of the rapid elimination of bromine following injections with sodium chloride.

PURPURA HEMORRHAGICA (ESSENTIAL THROMBOCYTOPENIA): TREATMENT BY THE MERCURY VAPOR LAMP. E. TOLSTOI, J. A. M. A. **89**:370 (July 30) 1927.

Tolstoi reports three cases of purpura hemorrhagica in which ultraviolet radiation was of no value. He was induced to try this treatment by the favorable results reported by Sooy and Moise; he was unable to corroborate their results.

NEED FOR RESEARCH IN THE TREATMENT OF EPIDERMOPHYTOSES. J. H. MITCHELL, J. A. M. A. **89**:421 (Aug. 6,) 1927.

Mitchell pleads for an intensive attack on this problem. The therapeutic advances in epidermophytoses of the hands and feet in the past few years are almost negligible. He feels that routine microscopic as well as cultural examinations should be made in all cases. Sabouraud's original postulates in regard to cultural and clinical uniformity of the various species are now either doubted or rejected. Among the newer therapeutic suggestions, Mitchell has found the thymol-cinnamon combination of Myers and Thienes of value. Trichophytin appears to be valueless both therapeutically and diagnostically.

BISMUTH IN THE TREATMENT OF CONGENITAL SYPHILIS. C. S. WRIGHT, J. A. M. A. **89**:424 (Aug. 6) 1927.

Wright reports fifty-four cases. Courses of sixteen injections (each 5 mg. per kilogram of body weight) were given, with an interval of one month between courses. The maximum dose was 100 mg. of the drug. In three cases in which there were active skin lesions, these manifestations disappeared rapidly. Forty-seven patients had late congenital syphilis, and of these, forty-three had positive Wassermann reactions after considerable treatment with neoarsphenamine. About half of these patients had a negative Wassermann reaction after bismuth therapy. Wright found bismuth satisfactory in four cases of interstitial keratitis. The drug was well tolerated and is recommended as the chief adjunct of the arsphenamines in the treatment of patients with congenital syphilis.

EPITHELIOMA OF THE PENIS: REPORT OF FIVE CASES. C. H. deT. SHIVERS, J. A. M. A. **89**:446 (Aug. 6) 1927.

In each case, irritation seemed to play a rôle in the development of the malignancy. All the cases were of the squamous cell type. In two cases partial, and in three, total amputation of the penis was performed. In three cases, cure was obtained, while in two, recurrences or metastasis occurred.

THE TREATMENT OF CENTRAL NERVOUS SYSTEM SYPHILIS. J. E. MOORE, J. A. M. A. **89**: 588 (Aug. 20) 1927.

The paper is based on the author's experience with 261 patients with neurosyphilis followed for an average period of four years. A thorough study of the patient is essential; the type of neurosyphilis he presents should be identified, and appropriate treatment for this type instituted. Schematic treatment, however, is not always possible, and in many cases therapy must proceed by the trial and error method. Moore's therapeutic scheme is as follows: (*a*) for "minimal" neurosyphilis, routine antisyphilitic treatment; (*b*) for early meningeal and late meningovascular neurosyphilis, an intensification of routine treatment; for serologically resistant cases of these types, tryparsamide, malaria or both; (*c*) for selected cases of tabes, intraspinal treatment; (*d*) for general paralysis, tryparsamide, malaria or both, and (*e*) for optic atrophy, intraspinal treatment.

With these methods of treatment, it has been possible in an ambulatory clinic to obtain 62 per cent of excellent or good combined clinical and serologic results in early meningeal neurosyphilis; 59 per cent in late meningovascular neurosyphilis and 31 per cent in tabes. Remissions have been secured in 57 per cent of a small series of patients with early general paralysis and 40 per cent of satisfactory results in early primary optic atrophy. In the latter condition intraspinal therapy offers the only means of arresting the process. Intraspinal therapy in tabes is of decided value.

A Study of Two Hundred and Two Cases of Congenital Syphilis. A. B. Cannon, J. A. M. A. **89**:666 (Aug. 27) 1927.

Cannon's series revealed that sixty cases represented the first pregnancy, with a decreasing number for succeeding pregnancies. This is contrary to the usual teaching as expressed in Deday's law. The average age at which symptoms developed for which the patient sought medical attention was between 10 and 15 years. The majority of the patients were at least of average intelligence. The cutaneous manifestations were similar to those in adults with secondary syphilis. Interstitial keratitis was the most frequent clinical manifestation, being present in seventy patients. The most common defect was dental abnormalities; thirty-three had mulberry molars and twenty-one Hutchinson's teeth. There were sixteen cases of neurosyphilis; about 10 per cent of the series showed positive spinal fluid tests. The patients were treated on the continuous plan with neoarsphenamine, mercury and potassium iodide. In neurosyphilitic cases intraspinal treatment was carried out with satisfactory clinical results in many instances. Fourteen cases of syphilitic nephritis and four of syphilitic meningitis were encountered. All were fatal.

A Study of the Skin in Five Hundred Cases of Diabetes. A. M. Greenwood, J. A. M. A. **89**: 744 (Sept. 3) 1927.

Greenwood's study was instigated by the reports of recent years on the association of hyperglycemia and certain disorders of the skin. It appears from the present study that the incidence of diseases of the skin is higher among diabetic than among nondiabetic persons. The person with diabetes shows no greater incidence of fungus infections, but the condition is potentially more serious. A diabetic person with dry skin is twice as liable to infection as a person with normally moist skin. Pruritus, both local and general, was more frequent than in the general run of patients. The author assumes that hyperglycemia may be a factor in certain diseases of the skin.

Michael, Houston, Texas.

The Place of Erythema Nodosum in Medicine. G. F. Walker, Brit. J. Dermat. **39**:241 June) 1927.

Walker discusses the place of erythema nodosum in medicine under four headings, citing references to the literature and cases of his own to demonstrate his point under each heading. Under part 1, "Erythema Nodosum Has No Connection With Rheumatic Fever," he refers to the close alliance to rheumatism described in almost every textbook; he believes this theory had its origin in the paper by Stephen Mackenzie in 1886. Walker says that after reading the literature thoroughly he cannot find that any close association has ever been established between erythema nodosum and rheumatic fever to the exclusion of other etiologic factors. Under part 2, "Is Erythema Nodosum the Rash of an Acute Specific Infectious Disease?," he gives credit to Lendon for first calling attention to this conception in 1890, and in 1905 in his essay "Nodal Fever." Walker believes that from the general mass of cases of erythema nodosum such a precise clinical entity—nodal fever (Lendon's disease)—emerges. Nodal fever must be disentangled from a nodose variety of erythema multiforme, and from the erythematous nodosities occurring in various phases of tuberculosis. Walker says that he believes that fully half of all cases of erythema nodosum are nodal fever, and that nodal fever is an acute specific, mildly infectious disease. Its incubation period is about fourteen days. The prodromal period may be so intense as to simulate typhoid fever; the exanthem is precise and uniform, relapses are common, and the convalescence may be fraught with danger. The disease is more common in spring and autumn; is common up to early adult life, and attacks women rather than men. He discusses the question of the infectious nature of the malady citing Lendon's paper and other references. Under part 3, "The Association Between Erythema Nodosum and Tuberculosis," he cites cases reported in the literature in which

tubercle bacilli were demonstrated, or in which the patient showed signs or symptoms usually associated with tuberculous involvement. He refers to the suggestion, by Abt, that erythema nodosum may well be an acute specific infectious disease, with a particular likelihood that in the postfebrile debility tuberculosis may be established. Under part 4, "Erythema Nodosum. Neither Tuberculous Nor Febrile," he takes up a group of miscellaneous cases of the disease; (*a*) cases in which erythema induratum begins as an indolent relapsing erythema nodosum; (*b*) cases grouped among the cutaneous manifestations of syphilis; (*c*) cases associated with septicemia; (*d*) the nodose form of erythema multiforme; (*e*) the isolation of a diphtheroid bacillus as the causal agent by Rosenow; (*f*) erythema nodosum as a phenomenon of anaphylaxis; (*g*) provoking of a rash superficially resembling erythema nodosum by some drugs, and (*h*) cases in which multiple pathologic changes are present.

He submits the place of erythema nodosum in some such grouping as follows: erythema multiforme, erythema nodosum in hypersensitiveness, erythema nodosum as the rash of nodal fever, erythema nodosum in tuberculous infection and erythema nodosum as the beginning of Bazin's disease. The papulonecrotic tuberculids.

A complete bibliography follows the article.

A Simple Form of Sudorimeter. P. B. Mumford, Brit. J. Dermat. **39**:256 (June) 1927.

An apparatus is described, with schematic drawing, that measures the loss of moisture from the hand. The results from a series of about 100 examinations indicate that the total loss of moisture from the hand in the average healthy person lies between 0.30 Gm. and 0.60 Gm. per ten minutes in man, and rather lower in woman.

Two Cases of a Chilblainy Condition of the Legs Somewhat Resembling Erythema Induratum; Also A Case of Erythema Induratum for Comparison. F. Parkes Weber, Brit. J. Dermat. **39**:259 (June) 1927.

The histories of three cases occurring in young women are presented. In his conclusions, the author mentions the part of modern dress in the causation of these chronic chilblain conditions, and the fact that perhaps these chronic conditions of the legs may, in especially predisposed subjects, favor the development of true (tuberculous) erythema induratum.

Note on Two Cases of Lichen Nitidus. G. B. Dowling, Brit. J. Dermat. **39**: 285 (July) 1927.

Dowling refers to the recent report on this condition by Barber and describes two of his own cases. Illustrations accompany his descriptions, two photomicrographs being used in his first case. This case is that of a young married woman, aged 32, who had a mixed variety of lichen nitidus containing patches on the extensor surfaces of the knees and right elbow, with discrete papules on other parts of the body, chiefly on the shoulders. The eruption was mildly pruritic, and the Pirquet test was negative. The second case occurred in a woman, aged 66, who was seen in September, 1926, with lichen planus. A year previously she had had a similar eruption. The last attack cleared up in about two months. Three months later she returned with an eruption of lichen nitidus papules on the forehead, neck and forearms, unaccompanied by irritation. Dowling discusses the possibility of lichen nitidus eventually proving to be one of the many clinical varieties of lichen planus; he refers to a case reported by Pels as miliary lichen planus, and points out a resemblance to lichen nititus in Pels' case.

ENDOTHELIOMA IN AN INFANT. ERIC I. LLOYD, Brit. J. Dermat. **39**:289 (July) 1927.

A case is described in a boy, aged 15 months, who at birth presented a small, hard, white spot in the left suprascapular region that gradually increased in size until the last few months, when it progressed rapidly. Local excision was performed, and the sections were diagnosed as probable endothelioma. An enlarged axillary lymph gland removed later showed metastases similar to the original tumor. Three photomicrographs accompany the article.

RECURRENT ONYCHOPTOSIS OCCURRING AS A FAMILY DISORDER. W. J. OLIVER, Brit. J. Dermat. **39**:297 (July) 1927.

Three cases are reported, the first one being observed by the author, and the notes on the other two being supplied by the parents. The first case is that of a healthy boy, aged 12, whose big toenail was involved and the second case is that of the boy's father, aged 42, who first shed his great toe-nail at the age of 36, and then annually for three years. In the third case, the brother, aged 14, shed both toe-nails when 12 years of age. Reference is made to cases reported by Montgomery and by Hutchinson.

XANTHOMATOSIS CUTIS AND HYPERCHOLESTEROLEMIA. J. T. INGRAM, Brit. J. Dermat. **39**:335 (Aug.-Sept.) 1927.

Ingram reviews the literature on blood cholesterol in xanthomatosis and on the pathogenesis of hypercholesterolemia and gives a detailed report of a girl, aged 11. The effect of insulin in hypercholesterolemia is discussed. The patient had xanthoma tuberosum with deposits in the tendons and a blood cholesterol of 430 mg. per hundred cubic centimeters. There is a description of miscroscopic sections of the tumor masses that were excised. There was no apparent abnormality of hepatic, pancreatic or renal function. The blood cholesterol was not affected by administration of insulin. The author suggests that hypercholesterolemia in this case was due to a primary metabolic disorder. Treatment of the patient followed the lines of local applications, general measures having failed.

NOTES ON A CASE OF KERATOSES AND FIXED PIGMENTARY DEPOSIT FOLLOWING QUARTZ LIGHT THERAPY. K. G. COLQUHOUN, Brit. J. Dermat. **39**:346 (Aug.-Sept.) 1927.

A case is described in a man, aged 27, who received treatment by an air-cooled mercury vapor lamp twice weekly for approximately eighteen months for bronchial catarrh. He developed multiple keratoses and fixed macular pigmentation as a result of the treatments. The picture conformed clinically to what is known as "solar keratoses." The keratoses were definitely fewer in number, flatter and more discrete when seen at a later date and suggests the amelioration of the pathologic condition when the causative agent has ceased to operate. The case further suggests the possibility of inciting cutaneous cellular reaction to the point of keratosis formation and fixed pigment deposit, and the presence of a latent period between the removal of the exciting factor and the initiation of skin changes.

THE DETECTION OF RINGWORM HAIRS ON THE SCALP BY THEIR FLUORESCENCE UNDER ULTRA-VIOLET LIGHT. A. C. ROXBURGH, Brit. J. Dermat. **39**:351 (Aug.-Sept.) 1927.

This method consists of examining the head in a darkened room by the light from a mercury vapor or tungsten arc lamp passed through a piece of Wood's glass. Wood's glass is dark violet and contains oxide of nickel. It allows only a very little red and visible violet and some long ultraviolet rays to pass. Normal hair and skin show very slight flourescence under this light, the

finger-nails react more brightly, but hairs infected with ringworm give a brilliant fluorescence with a greenish light. This enables one easily to identify small groups of infected hairs or even single stumps on an otherwise healthy scalp. The appearance becomes pathognomonic only when the hairs are infected. The author finds the method of value in ascertaining whether epilation after thallium acetate or roentgen-ray treatment is complete, or whether a few ringworm stumps still remain; it is also of value in deciding whether the brother or sister of a patient with ringworm is infected.

MEETING OF THE SECTION ON DERMATOLOGY OF THE ROYAL SOCIETY OF MEDICINE HELD MARCH 17, 1927. Brit. J. Dermat. **39**:354 (Aug.-Sept.) 1927.

At this meeting, Prof. W. E. Dixon (Cambridge) gave a short address on thallium, giving an explanation of the results of experiments in which alopecia was produced by thallium in man and animals. He explained that when thallium was injected in moderate doses in man or animals, the plain muscle throughout the body immediately relaxed. It also had a remarkable action on the autonomic nervous system acting in much the same way as strychnine by allowing reflexes to pass through the spinal cord more easily than usual. Normal stimuli were exaggerated under the use of thallium. There was no other substance which acted like thallium in producing the characteristic alopecia in man and animals. All animals, with the possible exception of the guinea-pig, lost their hair. Small doses of thallium stimulated the growth of hair. Experiments on rabbits were shown demonstrating these points. Sections of the area of alopecia in rabbits showed an almost normal epidermis; there was increased density of fibrous tissue in the dermis, but the sebaceous follicles were normal; collections of epithelial cells were smaller than usual, but the dividing cells showed nothing abnormal. Interruption in the normal growth of hair was due to the failure of the process of transition from the large polygonal cells to the stratified cells forming the hair. The nerve cells on the course of the fibers in the autonomic system were mainly affected. The association of alopecia, at times with nervous phenomena in the patient offers evidence that injury of the nerve might produce alopecia, and that the autonomic system might have a part in its causation. It was only after absorption that thallium affected the hair. Thallium was found in almost every tissue in the body in postmortem examination of animals that had been treated with it; the skin, however, retained less thallium than any other tissue. Animals fed with thallium and then killed showed a deficiency of epinephrine in the suprarenal glands.

WIEN, Chicago.

CHRONIC TRICHOPHYTOSIS OF THE SKIN AND NAILS IN ALL MEMBERS OF A FAMILY, WITH INVASION OF THE MUCOSAE, LYMPH NODES AND BONE. A. PELÉVINE and N. TCHERNOGOUBOFF, Ann. de dermat. et syph. **8**: 403 (July) 1927.

In a mother, two sons and a daughter, the nails were infected, and in the children wide areas of the skin were involved. In one there was granuloma formation, and sinuses were found leading to foci of infected bone; infected lymph nodes were present. In the daughter the organism was recovered from the vagina, and in one son the oral mucosa was infected. The children's nails had been infected soon after birth, the appearance of the generalized cutaneous involvement having been delayed. The nails were considered to have been the means of spreading the infection. A vaccine acted favorably.

OBSERVATIONS CONCERNING THE PRECEDING WORK OF PELÉVINE AND TCHERNOGOUBOFF. R. SABOURAUD, Ann. de dermat. et syph. **8**:420 (July) 1927.

Sabouraud stresses the fact that the parasite reached the derma through the follicle walls and not through the surface epidermis. Granuloma formation has been observed previously in trichophytic infections, although not by Sabouraud. He believes that *Trichophyton violaceum* is likely to produce unusual lesions in various locations.

PAROXYSMAL ACRODYNIA OF INFANCY. PIERRE BÉZY, Ann. de dermat. et syph. **8:** 425 (July) 1927.

A typical case of acrodynia is described, in a child aged 4 years.

MELANOCARCINOMA IN XERODERMA PIGMENTOSUM. J. NICOLAS, M. FAVRE and D. DUPASQUIER, Ann. de dermat. et syph. **8:**457 (Aug.-Sept.) 1927.

This case, occurring in a woman, aged 30, is described in detail, with a report of the necropsy observations. In addition to the usual syndrome, with the formation of basal cell epitheliomas, melanocarcinomas developed with widespread visceral metastases. The patient's oldest brother also was said to have had xeroderma pigmentosum.

Five other cases found in the literature are described, and eight others are briefly cited.

INVESTIGATION OF SPIROCHETES IN THE SYPHILITIC CHANCRE DURING THE COURSE OF SPECIFIC TREATMENT. MILAN KITCHEVATZ, Ann. de dermat. et syph. **8:** 473 (Aug.-Sept.) 1927.

In twenty patients, chancres were examined for spirochetes every few days during a course of injections of neoarsphenamine and of mercury salicylate. The neoarsphenamine was given weekly in dosage increasing from 0.15 Gm., and the salicylate was injected twice a week. The microscopic observations were carefully checked by subsequent examinations, and it was found that in eight cases the spirochetes disappeared within four days; in three cases they persisted for twelve, twenty-two and twenty-eight days, respectively, and in the remaining nine their complete disappearance could not be demonstrated.

INTENSE PRIMROSE DERMATITIS OF THE BULLOUS TYPE. P. GIACARDY, Ann. de dermat. et syph. **8:**485 (Aug.-Sept.) 1927.

In this case, the eruption had recurred every spring, for three years, always fading in August. The symptoms were unusually severe, with large bullae and hyperkeratosis. *Primula obconica* was the causative factor.

SUPRASYPHILITIC LEUKOPLASIA OF THE UTERUS AND CANCER. P. MÉRIEL, Ann. de dermat. et syph. **8:**489 (Aug.-Sept.) 1927.

Mériel cites the literature and describes a case he observed in a woman, aged 26. He believes that syphilis is often the cause of uterine cancer, and that leukoplasia of the neck and even of the body of the uterus is frequently found in these cases. This leukoplasia is similar to that of the tongue, and appears to be largely due to syphilis. It seems to be the intermediate stage of cancer formation, and the author concludes that such an initial process plays an essential part in the pathogenesis of a large number of cancers of the uterus.

PARKHURST, Toledo, Ohio.

ACUTE SYPHILITIC MYELITIS. GIONA NARDI, Arch. ital. di dermat. e sifil. **2:**357 (April) 1927.

A case of early acute myelitis is reported in a man, aged 49. The nervous phenomena appeared five months after the chancre. The patient died in spite of intense antisyphilitic treatment.

THYROID GLAND IN DERMATOLOGY. M. ARTON, Gior. ital. di dermat. e sifil. **68:** 158 (May) 1927.

THYROID GLAND IN DERMATOLOGY. G. BERTACCINI, Gior. ital. di dermat. e sifil. **68:**233 (May) 1927.

THYROID GLAND IN DERMATOLOGY. B. SPARACCIO, Gior. ital. di dermat. e sifil. **68**:278 (May) 1927.

These three monumental contributions to the cutaneous manifestations of thyroid disfunction were read as a symposium at the twenty-third meeting of the Italian Dermatological Society, held in Rome in December, 1926. They contain a full review of the literature on the subject, and also experimental and clinical references.

PARATHYROID AND THE SKIN. G. MARIANI, Gior. ital. di dermat. e sifil. **68**: 288 (May) 1927.

Certain cases of dystrophic lesions of the nails, of alopecia and of scleroderma may be due to parathyroid defficiency causing a disturbance of calcium metabolism. In a case of epidermolysis bullosa studied by the author the parathyroid deficiency was proved.

PROFESSIONAL DERMATOSIS. R. CASAZZA, Gior. ital. di dermat. e sifil. **68**:671 (May) 1927.

Calcium cyanamide (Ca CN_2) is a fine, bluish-black powder used in agriculture in Italy. It produces toxic phenomena like tachycardia, dyspnea, congestion of the face and collapse. On the skin, it produces erythema, urticaria, eczematoid and bullous dermatitis and multiple and extensive sphaceloderma. Two cases of the latter are reported.

TREATMENT OF VULVAR PRURITUS. R. ARAYA and E. J. RONCORONI, Rev. méd. latino-am. **12**:1236 (June) 1927.

The authors recommend the practice of epidural injections in cases of vulvar pruritus. They inject 10 cc. of physiologic sodium chloride solution or the same amount of a 1 per cent procaine hydrochloride solution. In rebellious cases they use from a 5 to 10 per cent solution of alcohol once or twice a week. The epidural injection is also recommended in cases of anal pruritus and of anal fissure.

POSTARSPHENAMINE HEMORRHAGIC PURPURA. C. MADERNA, Riforma med. **43**: 704 (July) 1927.

The author reports a case in which complications followed arsphenamine therapy. The patient was treated successfully with intravenous injections of sodium thyosulphate and recovered. Purpura is due to the toxic action of the benzol element in arsphenamine, especially in patients with damaged livers and kidneys.

PARDO-CASTELLO, Havana.

DERMATOLOGIC ABSTRACTS

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

ROSACEA WITH ACCOMPANYING CONDITIONS. R. H. RULISON, Am. J. M. Sc. **174**:60 (July) 1927.

Complete gastro-intestinal examinations were made by Rulison on fifty patients suffering from rosacea. Two thirds of them were found to have a group of functional abnormalities occurring with sufficient uniformity to suggest that they form an essential part of the disease. These abnormalities were a neurotic tendency, subnormal weight, low blood pressure, poor muscle tone, faulty posture, visceroptosis, chronic constipation, spasticity of the large bowel, and gastric subacidity. Correction of these conditions together with local treatment resulted in rapid and lasting improvement in most cases.

Dermatitis Venenata in Rubber Tire Industry. D. E. H. Cleveland, Canad. M. A. J. **17**:695 (June) 1927.

Hexanitrodiphenylamine was responsible for the poisoning in Cleveland's case. Hexanitrodiphenylamine (known colloquially in the factor as "hex") is a power, readily conveyed through the air, which produces its effects on the face and on the parts covered by dusty sweat-soaked clothing.

Preservation of Herpes Virus. J. R. Perdrau, Brit. J. Exper. Med. **8**:167 (June) 1927.

A method is described by Perdrau for the preservation of the virus of herpes which is economical in animals and time saving, and which should be capable of preserving the virulence of the strain over considerable periods of time by limiting the number of animal passages.

Treatment of Ringworm by Thallium Acetate. S. J. Firth, Brit. M. J. **1**:1097 (June 18) 1927.

The dose of thallium acetate given by Firth was ⅛ grain (8 mg.) per 2¼ pounds (1 Kg.) of body weight, irrespective of age. The drug is easily soluble, and is made up in sweetened water and administered in a single dose. Constitutional symptoms were not observed after the administration of the drug, even in the case of one child who received a second administration six weeks later. Local treatment was not applied to the scalp. The shedding of hair commenced between the fourteenth and sixteenth days. The hair could then easily be pulled out without causing any discomfort, and it also came off in the caps. The shedding was in every successful case complete in from three to six days.

Thrombo-Angiitis Obliterans in Women. E. D. Telford and J. S. B. Stopford, Brit. M. J. **1**:1140 (June 25) 1927.

The interest in the two cases cited by Telford and Stopford lies not only in the rarity of the disease in women, but in the fact that the two patients illustrated very opposite types. In the first case the period between the onset of symptoms and the occurrence of total gangrene was only three months. In this patient the thrombosis had spread throughout the arm with such rapidity as to preclude entirely any successful attempt at collateral circulation. In the second case, the course was extremely chronic. The patient dated her symptoms six years back, and it was evident that the change for the worse had been very slow. While there was no pulse to be felt in either arm, it was plain that plenty of time had been given for the setting up of a good collateral circulation. There was, in fact, evidence that the history of this case was in reality very much longer than six years. The patient stated that at her last confinement both physician and nurse had been surprised to find that a pulse could not be found in either arm; this confinement had occurred no less than sixteen years before.

Epilation by Thallium Acetate. F. R. Curtis, Lancet **1**:1290 (June 18) 1927.

Using rats as experimental animals, Curtis found that epilation occurs constantly on the seventh or eighth day after the subcutaneous injection of a solution of thallium acetate equivalent to 7.5 mg. per kilogram of body weight. In all cases, the solution was freshly made immediately prior to injection. Solutions of thallium acetate, in similar doses, prepared and placed in sterilized ampules three months previously, were tested on a series of rats. Epilation occurred promptly on the eighth day after injection, thus showing that the date of the preparation is not of any moment. A point worthy of mention is the danger of giving a second dose too soon after the first dose.

TREATMENT OF IMPETIGO CONTAGIOSA BY ULTRAVIOLET RAYS. J. B. ELLISON, Lancet **1**:1343 (June 25) 1927.

Ellison gave ultraviolet ray treatment, using the Cooper-Hewitt direct air-cooled quartz mercury vapor arc lamp, in forty-one cases of impetigo contagiosa. He says that the rays have a slight, though definitely curative, effect. General exposure is safer and more efficient than local exposure at short range in this condition.

ULTRAVIOLET RAY TREATMENT OF CHRONIC ULCERATION. G. M. WAYCHOPE, Lancet **1**:1345 (June 25) 1927.

By means of ultraviolet irradiation, Wauchope obtained improvement under somewhat difficult circumstances in three cases of chronic ulceration of the legs when other methods employed had failed.

INFLUENCE OF IODOFORM ON WASSERMANN REACTION IN RABBITS. G. GJORGJEVITCH and M. PIEVATCHEVITCH, Compt. rend. de Soc. de biol. **96**:1006 (April 26) 1927.

Gjorgjevitch and Piévatchévitch injected twenty rabbits intravenously with an iodoform solution. After from ten to twelve injections, the Wassermann reaction became positive in the blood of the animals, and remained so for a few days. Subcutaneous injections of iodoform did not modify the Wassermann reaction. Traces of free iodoform were not present in the blood of the animals. Eleven patients with a negative Wassermann reaction were injected intravenously with increasing doses (from 0.05 mg. to 0.25 mg.) of iodoform in 3 or 4 cc. of water. The reaction became positive after from ten to twelve injections, totaling 1.5 Gm. of iodoform. The effect of subcutaneous injections was negative. A positive Wassermann reaction may occur in patients with soft chancres, treated for long periods by iodoform.

ACTION OF RAYS ON LYMPHOID ORGANS. J. JOLLY, Le sang **1**:1, 1927.

Jolly studied the lesions caused by lymphoid tissues by short wavelength rays other than roentgen or gamma rays. Changes produced by the ultraviolet rays have heretofore been noted only in the skin and inaccessible membranes, such as the cornea and the lingual mucosa. Owing to absorption of the ultraviolet rays by the skin, the lymphoid organs appeared unaffected by the rays. In his experiments, Jolly exposed the inguinal glands and the thymus of animals to rays, after having incised the corresponding part of the skin. Under ultraviolet rays, the lesions in the thymus were limited to the cortical portion of the gland, directly exposed to the rays. There was pyknosis and nuclear necrosis in the thymocytes. The connective tissue remained intact. In the inguinal glands, the lesions were analogous to those in the thymus. The glandular follicles, especially the germinal centers, were involved. The character of the lesions and the latency period were the same as when roentgen rays were employed. In another set of experiments, rats were injected with erythrosin and then the thymus was exposed to sunlight. The animals were killed from twelve to twenty-four hours after the exposure. The lesions in the gland were the same as after exposure to ultraviolet rays. Changes in the thymus did not occur from exposure to sunlight without the previous injection, or from injection without exposure to sunlight. Chemical changes in the body fluids, caused by the fluorescent substance, were responsible for the lesions. Evidently, rays of short wavelength exert, besides a direct action, also a secondary toxic action, the character of which is yet unknown.

CONTAGION AND TRANSMISSION IN LEPROSY. G. ARCOS, Bol. del Hosp. Civil de San Juan de Dios **1**: 19 (Jan.) 1927.

Arcos reviews, in a philosophic spirit, his long experience with leprosy. At the Pifo leprosarium, among 150 patients, he has not seen a single case of conjugal contagion. In about 66 per cent, inheritance may have a relative importance, as there are leprous parents, brothers, sisters, or cousins in the family. On the other hand, lepers may have healthy children. Some patients claim they have never been in contact with lepers. A couple, both leprous before marriage, have six children affected with the disease. Dr. Echeverria, of the same institution, quotes several cases of lack of contagiousness, among them that of a widow of two lepers, in succession, who lived among the sixty-five patients in the asylum and remained well. The rôle of insects seems very doubtful. Several patients who contracted itch from lepers months ago have not had any symptom or sign of leprosy. While house flies are prevalent in the leprosarium and carry acid-fast bacilli and contaminate food, there has not been a single case of fly-transmitted disease either among the employees or in the neighboring village. The inoculations made, for instance, in Colombia, are without value, as the fifteen children lived at leprosariums and were the offspring of lepers. Arcos has inoculated guinea-pigs and rabbits with leprous products, blood or macerated tubercles. The result was negative, although the tissues inserted in the peritoneum persisted and contained bacilli until their final elimination. He repeated Reenstierna's experiments on a macacus; after forty days a node developed, which contained bacilli. Morphologically, these were identical with those of the inoculated leproma, but the node regressed slowly and at the end of a year not a sign remains. The disease attacks the genital organs and may cause impotence and sterility. If women become infected before puberty, they never menstruate. There must be some unknown carrier of the disease or Hansen's bacillus may be a mere saprophyte or a modality of the pathogenic germ.

NEUTRAL BISMUTH PHOSPHATE IN SYPHILIS. R. SOUZA LOPES, Brasil-Medico **41**:417 (April 30) 1927.

Among the advantages claimed by Souza for neutral bismuth phosphate are its stable composition and content of bismuth, its insolubility and non-toxicity, its slow absorption and painless injection. The combination with phosphorus is also of advantage, because of the tonic value of the latter.

POSTOPERATIVE IRRADIATION OF CARCINOMA OF THE BREAST. W. ANSCHUTZ, Beitr. z. klin. Chir. **139**:25, 1927.

Anschütz insists that the value of postoperative irradiation of carcinoma is seen only in a selected group of cases. He explains the statistics of the Kiel University clinic. Small, frequently repeated doses—two thirds of the erythema dose, three fields, ten or eleven irradiations distributed over two years—have proved more efficacious than Wintz's intensive method.

BLANCHING PHENOMENON. M. WINKEL, Deutsche med. Wchnschr. **53**:963 (June 3) 1927.

Winkel observed white patches on the skin in two scarlet fever patients. They were localized on sites where furuncles had previously existed. In a third case the scarlet fever rash was absent around superficial scars left by chickenpox.

HYPERSENSITIVENESS TO RUBBER. G. STERN, Klin. Wchnschr. **6**:1096 (June 4) 1927.

Stern's patient suffered from urticaria and Quincke's edema. After six months it was discovered that the condition was due to hypersensitiveness to the rubber contained in a dental prosthesis.

INSUFFICIENTLY TREATED SYPHILIS IN CHILDREN. F. STERN, Klin. Wchnschr. **6:** 1235 (June 25) 1927.

Six children who had been insufficiently treated for syphilis from three and one-half years to twelve years before were reexamined by Stern. Only two of them showed any sign of syphilis.

"REGRESSION OF SYPHILIS AND ARSPHENAMINE."—H RUGE, Klin. Wchnschr. **6:**1237 (June 25) 1927.

Ruge attributes the regression of syphilis in the German navy to an energetic compulsory individual prophylaxis. Jadassohn critizises his figures and demonstrates on them the probability of another factor—apparently arsphenamine. The relation of the frequency of gonorrheal to that of syphilitic infections was 5:2, while it is 5:1.3 at present. He is strongly in favor of personal prophylaxis, although he admits that it may in some cases cause infection to be overlooked.

PRIMROSE DERMATITIS. W. EITEL, Med. klin. **23:**837 (June 3) 1927.

Eitel believes with the botanist Ulbrich that only about 6 per cent of people are hypersensitive to the primrose. He recommends washing the hands and face with a solution of from 1 to 2 tablespoonfuls of sodium bicarbonate in 1 liter of water in prevention as well as in treatment of the dermatitis.

Society Transactions

BROOKLYN DERMATOLOGICAL SOCIETY

Regular Meeting, Jan. 17, 1927

JOHN C. GRAHAM, M.D., *Chairman*

PSORIASIS. Presented by DR. BERKOWITZ.

S. O., a girl, aged 5½ years, had erythematous, scaly lesions on the distal half of the fingers and toes of one year's duration. On the palms and soles were several sharply defined, scaly lesions varying in size from that of a pea to that of a bean, and one was about the size of a half dollar. The nails showed horizontal striations and some pitting, and the ends of the nails were slightly elevated owing to scaling of the nail-beds. This patient was shown because of the localization of the lesions.

DISCUSSION

DR. THRONE: Before making a diagnosis of psoriasis, the child should be examined carefully for possible mycotic infection.

DR. FRANK: I do not think this is a case of psoriasis. There are no lesions on the rest of the body. I think it is some type of ringworm infection.

DR. CHARGIN: I think it is a case of psoriasis. It is a papular eruption, discrete and in patches, located on the palms and soles. The papules show scales, and most of the scaling is like that observed in psoriasis. The nails are affected. One of the nails in particular is definitely pitted. The chronicity of the condition is in favor of psoriasis. The points enumerated favor psoriasis as the likely diagnosis, despite the fact that there are no other lesions on the body. Such an occurrence is not rare.

DR. ABRAMOWITZ: This child shows only palmar and plantar involvement, which is unusual in children. The scalp is clean, and the involvement of the nails is not typical. I think an eruption of this type should be observed further. An examination for tinea should be made and likewise a blood test. By way of treatment, half strength Whitfield's ointment may help.

DR. ALTMAN: I have never seen a case of psoriasis on the hands and feet alone. On the hands the eruption looks like that in tinea.

DR. GIVENTER: I would suggest giving the child therapeutic tests. If Whitfield's ointment clears up the lesions, the condition probably is tinea.

DR. GAUVAIN: I agree with the diagnosis. I have seen several cases of psoriasis of the body in which the lesions on the hands and feet were identical with these. It is true that this is the first time I have seen lesions on the hands and feet alone. However, cases of psoriasis of the scalp alone are seen. In a later attack the lesions will probably be found on other parts of the body.

DR. RULISON: This child shows marked involvement of the nail-bed of some fingers. I asked the mother whether there had ever been any pus, and she said there had not. I cannot think of a case of ringworm involving the nails and producing paronychia such as this child shows that did not at some stage of the disease present a purulent discharge. I agree with the diagnosis of psoriasis.

DR. WALZER: Dr. Rulison has made some of the points that I expected to make. Psoriasis is a protean disease, and can simulate a great many diseases. The lesions on the back of the hands, on which the skin is not so thick, are not those of tinea, with their central involvement and color. The involvement of the skin at the junction of the nail with the skin is certainly not typical of tinea, since, when tinea does involve the nail, it is from the edge of the nail inward. The scaling is especially noticeable as typically psoriatic, and the color of the lesions is that of psoriasis.

ECZEMA (?). Presented by DR. ABRAMOWITZ.

Mrs. R. F., a native-born widow, aged 31, had had measles as a child; an appendectomy and a gynecologic plastic operation had been performed 3 years previously. One year later, an eruption broke out on her hips and spread to the rest of her body. She was treated at Philadelphia and at Johns Hopkins Hospital for psoriasis. She was also seen in New York at the Bellevue College Clinic. The eruption disappeared completely under exposure to the sun. At the Vanderbilt clinic three months before presentation, her condition was diagnosed as parapsoriasis, and a biopsy, taken from one of the lesions was in favor of that diagnosis. She was later admitted to the Gouverneur Hospital for acute catarrhal jaundice and was under the care of Dr. M. Packard, who referred her to the presenter for diagnosis of her skin disease. She had had considerable scaling on her scalp since the rash appeared, but frequent washing with soap preceded by oil and occasionally benzine kept her scalp almost clean. At presentation there was a small scaly lesion on the right ear. The eruption was also present on the extensor surfaces of the upper extremities, and consisted of vesicular and scaly lesions varying in size from that of a quarter to that of a half dollar. This was most marked in some of the newer lesions that were appearing on the shoulders and chest. The eruption also occupied the buttocks and the outer side of the lower extremities, and consisted of irregular, palm-sized, lichenified, scaly patches, rather sharply outlined. The scaling was fine and resembled that in psoriasis, but most of the lesions showed some vesicles, some thickening of the skin and abrasions from scratching. The patient was definite in stating that the lesions she had itched a good deal and that they became moist when the itching was most marked. The smooth, slightly scaly patches on the legs, nevertheless, reminded one of parapsoriasis. Four Wassermann tests were negative. Skin tests with food and foreign proteins were negative. Her catarrhal jaundice had improved. She was being treated with ultraviolet rays and receiving injections of sodium cacodylate, and a 10 per cent solution of silver nitrate was being applied to the eczematous patches. Calamine lotion was being used for the itching. She was improving under treatment.

DISCUSSION

DR. RULISON: I am rather skeptical about the diagnosis of parapsoriasis. I would rather accept that of psoriasiform eczema. The facts that there is itching and that the eruption has cleared up and reappeared argue against parapsoriasis.

DR. CHARGIN: I do not think that we are dealing with parapsoriasis. The papular character of the eruption, the itching, the infiltration, the fact that the eruption comes and goes, the color and location all speak against parapsoriasis. I think a possible diagnosis is eczematized ringworm.

DR. THRONE: I cannot see any signs of parapsoriasis. Of course, in a case of this kind it is difficult to make a diagnosis since the lesions have been

treated. There seems to be a certain amount of dermatitis that might have been caused by local applications, but in addition there seems to be an infiltration which is entirely foreign to my idea of parapsoriasis. I believe the diagnosis lies between an eczema and a possible prefungoid stage of granuloma fungoides.

DR. ABRAMOWITZ: This patient was rather carefully studied from a general medical standpoint while she was in the hospital, but nothing definite was found.

LUPUS ERYTHEMATOSUS. Presented by DR. WALZER.

A. G., a school girl, aged 16, with no familial history of tuberculosis, had an eruption of seven months' duration, which began on the face and index finger of the left hand, and spread to the scalp, ears, shoulders, arms, back and chest. The lesions were large, sharply defined, erythematous superficial areas. The duct openings were not conspicuous, and the scaling was slight. On the face, both within and outside of the involved areas, were numerous atrophic spots the size of a millet seed, and on the scalp were areas of alopecia. There were no constitutional symptoms.

DISCUSSION

DR. CHARGIN: I am in favor of the diagnosis of acute lupus erythematosus. The superficial character of the lesions, which are erythema multiforme-like in character and the involvement of the face, chest and fingers favor the acute rather than the disseminated variety. Gold preparations should be administered in small doses.

DR. ABRAMOWITZ: It would be interesting to try gold therapy in this case.

NOTE.—After presentation of this patient she was seen in the Gouverneur Hospital in a state of coma. The rash was about the same, an erythema being also present on the trunk. She had not received any internal medication. A spinal tap revealed a meningococcus meningitis. About two weeks later she died in the hospital from an acute pulmonic infection, but it is not known whether this was tuberculous or not.

LEUKOPLAKIA BUCCALIS. Presented by DR. WALZER.

M. B., a man, married, aged 43, had had a urethral discharge twenty years previously, which had been cured in one month, but he said that he had never had syphilis. He had had two children, one of whom had died in infancy of an unknown cause. For one year the patient had had numerous pinhead to pea-sized, thin, bluish white, noninfiltrated patches on the mucous membranes of the cheeks, lower lip and gum. The tongue, which was infiltrated, was of the geographic type, showing the indentations of the patient's artificial teeth, and also presented many of the bluish white patches. The Wassermann reaction was four plus.

DISCUSSION

DR. ABRAMOWITZ: I think the condition is syphilitic. I have never been able to find a good method of treatment that is painless and permanent in leukoplakia.

DR. HECHT: I would treat the patient for syphilis and not worry about carcinoma.

DR. CHARGIN: I am certain that there is no satisfactory method of removing leukoplakia. The patient should receive mild antisyphilitic treatment. The active syphilitic process, if present, will disappear; the leukoplakia will remain. Mouth hygiene is important; decayed teeth and cavities should be looked after.

and mild mouth washes should be employed. I think leukoplakia is always precancerous. The patient should be cautioned against irritating the lesions by hot foods, smoking, spices, etc.

DR. WALZER: I do not believe any method of treatment will cure leukoplakia. Whether the man is syphilitic or not, the leukoplakia will remain in spite of antisyphilitic treatment. The only time treatment would be indicated would be if there were ulcerations developing on the leukoplakia. But in this case, the man has an extensive leukoplakia, and to attempt to remove it by any means would be wrong. With a positive Wassermann reaction, he is entitled to antisyphilitic treatment, although the leukoplakia will remain.

DERMATITIS DUE TO FEATHERS (?). Presented by DR. ABRAMOWITZ.

M. L., aged 64, a Jewish pedler, born in Ireland, who had lived in the United States for forty-four years, had had itching of the scalp and face for five years. For five months he had had an eruption of marked severity on the extremities and a slight eruption on the torso. On the backs of the hands and on the lower parts of the legs, where it was most severe, there were sharply circumscribed, weeping and scaly patches varying in size from that of a silver dollar to that of a palm. In addition, the legs showed localized hemorrhages. Itching was severe in all areas. In one clinic that he attended, a large amount of blood sugar was found, and he was given eight injections of sodium thiosulphate, without improvement. It was discovered from the patient's history that he was more uncomfortable after going to bed. He was advised to change his sleeping quarters and to substitute ordinary blankets for his feather covering. Since this has been done, he has been more comfortable at night. His toleration for feathers will be tested as soon as the skin becomes normal.

DISCUSSION

DR. WALZER: I saw this man some time ago for an exudative dermatitis of both legs and a pruritus and erythema of the thighs and lower part of the abdomen. I could not obtain any history of hypersensitivity in either the patient or any member of his family, and so I treated him locally. He improved, but the condition recurred in a somewhat milder form. I believed he had a dermatitis of external source. To call this condition an "anaphylactic dermatitis" because the condition cleared up, or almost did, after the patient had ceased to use a feather bed, and after he had received local treatment, is not to my mind correct. He has not been tested for sensitiveness to feathers; but even if he were, I will venture to predict that he will not give any definitely positive reactions. I say this because unless this man had involvement of the mucous membrane together with his skin involvement, he will not give any positive reactions to feathers by the intradermal or scratch test.

DR. THRONE: I treated this man once. A diagnosis of chronic eczema was made, and he was given sodium thiosulphate. The blood was examined for sugar and chlorides. The amount of sugar in the blood was high. We have found in treating patients who have eczema with sodium thiosulphate that it is almost useless in cases in which there seems to be an element of external sensitization. When this patient did not respond in a reasonable time to the injections, we felt there must be some external sensitization. I do not agree with Dr. Walzer that an adult cannot become sensitized to feathers. I have had cases in adults who got well after changing the pillows on which they were sleeping.

DR. ABRAMOWITZ: I am presenting this patient as having a possible case of anaphylaxis, using the term in a broad sense. I am not absolutely certain

that it is due to feathers, because I have not tested him yet. He has improved rapidly since his pillows and blankets have been replaced by other materials, more than the local treatment can account for. I have had similar experiences from previous cases of this type and also from those due to silk and pyrethrum insect powder.

E. Almore Gauvain, M.D., Secretary.

THE NEW YORK ACADEMY OF MEDICINE, SECTION ON DERMATOLOGY AND SYPHILIS

Regular Meeting, April 5, 1927

M. B. Parounagian, M.D., *Chairman*

Syphilitic Reinfection. Presented by Dr. Parounagian and Dr. Mason.

R. L., aged 39, when first seen on Nov. 9, 1921, had a large chancre on the left side of the lower lip, the scar of which persisted to the day of presentation. He also had roseola, and moist papules on the genitals and inner aspect of thighs, and the Wassermann reaction was four plus. He did not have a genital chancre.

He received eight injections of silver arsphenamine and fifteen of bichloride from November 9, 1921, to April 4, 1922. At the end of this course, his Wassermann reaction was two plus; a second course of six injections of arsphenamine and fifteen of mercury was administered from June 28 to October 17.

Since then, thirteen Wassermann tests were made from November 28 to October 25; all of them were negative. During this period the patient was entirely free from lesions or symptoms.

He returned for another Wassermann test on March 14, 1927, which was reported four plus, and the examiation revealed a generalized maculopapulosquamous eruption, palmar syphilids and the remains of a recent chancre on the prepuce. The roseola had been present since the beginning of March. He had not noticed the genital sore.

DISCUSSION

Dr. Goodman: I do not think that any case of reinfection presented until now fulfils all the rules and regulations for reinfection. It is necessary to establish first a primary infection beyond any possibility of doubt. This must be done by finding spirochetes in a chancre; the Wassermann test must first be negative and then positive. There must be enlarged regional glands. All this must occur in a patient who never had a previous infection.

The presence of the first infection having been satisfactorily proved, the patient must have had a thoroughly supervised course of treatment. Regression of the clinical lesions and the reversal of the positive Wassermann test to negatve should have taken place. There should be a subsequent period of freedom from clinical lesions of syphilis. The Wassermann test should have remained negative despite attempted provocation by injections of drugs of the arsphenamine group. Involvement of the cerebrospinal system must be excluded by suitable tests. This means that infection with syphilis was established and that as far as is known the patient was cured.

The patient under consideration must go through the cycle of infection in the proper order. He must have been exposed; the incubation time must have elapsed; the clinical lesion of chancre must be confirmed by bacteriology and the

Wassermann test at this stage must be negative. The second chancre must be on a surface other than within the same draining area of lymphatics as the first chancre. The patient should then have passed through clinical and serologic syphilis the second time, the same as the first.

If any links are lacking; if there is any break in the continuity of accumulated evidence; if one or another factors just set down cannot be satisfactorily met, it is my opinion that reinfection in syphilis has not been established. In the interest of scientific syphilology some such criteria must be advanced and adhered to.

DR. CHARGIN: If Dr. Goodman does not accept the case as one of reinfection, how can he explain the present eruption which, clinically at least, is clearly a maculopapular exanthem belonging in the first few months of syphilis? Surely, one does not obtain this type of eruption as a sequence to a chancre six years previously.

DR. RULISON: I am very much interested in this case, for I have been watching the cases presented from Bellevue for six or seven years and I cannot agree with Dr. Goodman in his position in regard to syphilis. It is true that there is not any absolute and final criterion of cure of syphilis, but the number of cases shown by the chairman of this section in which definite secondary lesions have appeared some years after verification of the original infection seems to represent definite progress in the treatment of syphilis. If one reads Fournier and other older authors on syphilis, it will be found that apparently reinfection or superinfection was uncommon. This case is of especial interest because there was a long period in which there was not any evidence of syphilis and positive Wassermann reaction and then frank secondary syphilis developed. I believe the old opinion must be revised; syphilitic patients are now cured, and reinfection is possible. The man did not reappear at the time of his second chancre, and did not have a dark-field and a negative Wassermann reaction; but the chairman has shown numerous cases in which all the criteria were presented, and the only question was the comparatively short period since the reinfection. In this case, the interval was six years. I think that the condition is syphilitic reinfection.

DR. ELLER: I think that today most men believe that early syphilis can be cured in patients who are treated in the first six months, and I do not see why the patient cannot be reinfected after a cure. Despite the fact that all the criteria have not been present in this case, I believe it is one of reinfection, and I believe that many others which Dr. Parounagian has presented here are cases of reinfection.

DR. LEVIN: I protest against the positive assertion made by Dr. Goodman in regard to cases of reinfection that have been presented before this section. It is true that this case does not satisfy all the criteria for a cure in syphilis, but most of these have been satisfied and it seems probable that this is a case of reinfection. Cases of true reinfection have been presented before this section and other dermatologic societies in this city. It should not be allowed to go on record that cases have been presented that were not proved to be reinfection. Several such cases have been seen and will continue to be seen. The fact that more cases of reinfection are seen than formerly may be explained by the fact that patients are receiving more and better treatment today than in previous years.

DR. PAROUNAGIAN: Sometimes spinal tests may be negative in the presence of marked clinical evidence of neurosyphilis.

If cases of this kind are not accepted as reinfection, one must admit that syphilis is an incurable disease and that reinfection cannot occur. However, I believe that syphilis is a curable disease, if the patient is treated early and properly, and that reinfection can and does take place.

EPIDERMOLYSIS BULLOSA ACQUISITA. Presented by DR. ABRAMOWITZ.

Miss L. M., aged 17, born in the United States, of Italian parentage, was a private patient of Dr. Oleynick's. She had always been in good health. There was no history of skin disease in the family, or of consanguinity in the parents. In October, 1926, the patient noticed for the first time erythematous patches on her arms, which eventually became bullous. After a time the bullae partly collapsed, partly opened and healed on application of a neutral ointment, leaving a temporary redness without scars. Since then the patient, and her house physician who referred the case, noticed that any external trauma, such as knocking, rubbing or pressing, after a short time (about two hours) provoked the formation of bullae at the place of the trauma.

As presented, the patient exhibited on her hands and arms a few bullae caused by recent traumas, and a few crusts and red marks as sequelae of former bullae. Occasionally, bullae were observed on the edges of the tongue. The patient also exhibited epidermal cysts on the ears. She had a hemoglobin content of 60 per cent.

DISCUSSION

DR. SCHEER: The case is interesting and typical. In addition to the usual lesions on the hands, the patient also has the small milia cysts so commonly found in this disease.

DR. WISE: I accept the diagnosis as presented. Not only is it a typical case, but it is also the most pronounced case of the kind ever presented before this section, and as far as my knowledge of the literature goes it is the most pronounced case of the "acquired" type of epidermolysis bullosa recorded.

In connection with this subject, Engman and Mook wrote that the elastic tissue is diminished in the congenital cases. In certain cases examined at the former dermatologic department of the Vanderbilt Clinic, elastic tissue was found to be normal in quantity and arrangement; in others, it was fragmented. It is hardly conceivable that the elastic tissue should undergo a change so late in life as in this patient, and that changes in the elastica are the cause of the bullae.

As to the treatment, I would suggest heavy doses of arsenic by intramuscular injections.

DR. BECHET: I do not wish to question the diagnosis, but the lesions seem almost too numerous for epidermolysis bullosa. The patient has various sized, violaceous plaques, probably the remains of bullae, and even a few papules. This is certainly suggestive of erythema multiforme of the bullous type, and I think that that diagnosis should be considered before epidermolysis bullosa is finally accepted.

DR. ABRAMOWITZ: I do not think there is any question about the diagnosis of epidermolysis bullosa. One does not see the peculiar cystic lesions on the ear in erythema multiforme.

PEMPHIGUS. Presented by DR. FELDMAN.

A. M., aged 17, a typist, had never had any serious illness outside of the usual diseases of childhood. About seven weeks prior to presentation, she developed sores in the mouth, and two weeks later the right eye became red and swollen. At about the same time a few blebs appeared on the skin. She presented herself for examination on March 20, 1927, and the following conditions were noted: In the oral cavity were broken down bullae on the tongue, buccal mucosa, pharynx and epiglottis, and there were several unbroken bullae on the lips. There was a bullous lesion on the sclera of the right eye. Several bullae were present on

the vulva. The skin presented a number of bullous lesions, but simultaneously with these there was an acute erythema. This served to cloud the diagnosis at first, but it was later discovered that the erythema was due to some form of medication used. The patient was seen again a week before presentation. At that time the lesions on the skin were practically gone and the lesion of the eye had disappeared. As exhibited, the condition in the mouth was somewhat improved; a new lesion was developing on the left sclera, and there was a new crop of bullae on the skin, most of which contained clear serum and were not associated with any signs of inflammation.

DR. OCHS: This case is rather exceptional on account of the youth of the patient, and I see no reason to doubt the diagnosis; the lesions in the mouth would lead me to suspect pemphigus, and when one sees the back, particularly the portion not touched by sitting on the chair, and finds the small bullae developing on a non-erythematous base and light and easily broken, I do not think any one would doubt the diagnosis of pemphigus. I would like to know the opinion of the members as to whether or not pemphigus is on the increase. Not many years ago it was rare. Today, I doubt if any dermatologist does not see a number of cases during the year.

DR. TRAUB: The condition is certainly pemphigus. A word about the erythema that has been mentioned. There are some cases of pemphigus in which there is definite erythema, and later bullae develop where the skin was previously erythematous. I have just had a typical case in the hospital, in which bullae invariably followed an erythema. As far as the increase in pemphigus is concerned, I have seen five or six cases in a short time; whereas before, that would have been a fair number to see in a year. I have presented a patient who had lesions only in the mouth until two or three days ago, when typical blebs appeared on the back.

DR. ABRAMOWITZ: I cannot say anything about the increase of these cases in the last few years, but I would like to call attention to an article in the *Archiv für Dermatologie und Syphilis* by an investigator who noted a series of cases in which there were diminished chlorides in the urine and an eosinophilia. The importance of this observation lies in the fact that this investigator drew prognostic conclusions, for in most of these cases the condition started with lesions in the mouth before any lesions developed on the body. It would be interesting to have such investigations made on the cases presented.

DR. CHARGIN: The work referred to by Dr. Abramowitz has been tested out in two cases at the Mount Sinai Hospital in the service of Dr. Goldenberg. Kartamischew, as may be recalled, is of the opinion that there is an "edema tendency" in the skin of pemphigus patients, and that this is indicated and could be determined by a study of the behavior of sodium chloride in the body. In the two cases referred to, Kartamischew's results could not be confirmed.

DERMATITIS DUE TO PICTORIAL PAPERS. Presented by DR. HOPKINS.

C. E., a white woman, aged 46, born in Austria, had had an eruption from time to time for nine weeks. The rash always appeared on Sunday night or Monday morning, and by Friday the condition of the affected skin was normal again. On the Monday previous to presentation there was a papulo-erythematous dermatitis on the flexor surface of both forearms, and a dark erythema of the face with edema of the eyelids. The patient was advised to avoid contact with the Sunday edition of her newspaper, and the condition did not recur.

DR. LEVIN: I have seen a similar case in my office. The patient was under treatment for erythema of the face and neck which recurred over a period of

years. All possible features for producing the dermatitis were ruled out. However, on close questioning, it was discovered that the patient was in the habit of reading the pictorial section of the Sunday paper, and would go to sleep with the paper over his face. The eruption was always worse on Mondays. Since he has refrained from reading the rotogravure section of the paper, the eruption has not returned.

GRANULOMA INGUINALE SIMULATING EPITHELIOMA. Presented by DR. CANNON.

M. B., a colored woman, aged 19, born in the United States, about ten months previous to presentation noticed a vaginal discharge and swelling of the labia majora. Three months later, a hard nodule, the size of a dime, developed on the left side, gradually broke down and ulcerated. The Wassermann reaction was positive, and the patient was given ten injections of arsphenamine and ten injections of mercury and potassium iodide for two months without improvement in her condition.

Examination showed the labia majora and minora greatly enlarged, especially on the right side, which measured about 4½ by 3 inches (11.4 by 7.6 cm.). The outer skin surface was smooth and hard. The opposing surfaces of the labia minora showed sites of ulceration about 2½ by 1½ inches (6.27 by 3.7 cm.) in diameter. The bases were irregular, consisting of granular, warty-like ulcerations, some reddish and some brown in appearance. The edges were rolled, elevated and hard. There was not any bleeding or adenopathy. The Wassermann test was negative. Urinalysis showed a faint trace of albumin.

The biopsy report disclosed epithelioma.

The patient had had ten injections of antimony potassium tartrate, with great improvement in the ulcer.

DISCUSSION

DR. POLLITZER: More care should be taken to send specimens for examination to pathologists who are properly qualified for skin work. Time and again the reports on pathologic specimens do not fit in at all with the clinical picture. The general pathologist usually knows but little about the skin. More care, too should be exercised in taking the specimens, as to the direction of the cut and the portion of the lesion from which they are removed.

DR. WOLF: It has been Dr. Cannon's good fortune in the past two years to present two cases in which there has been a discrepancy between the clinical and the pathologic diagnoses. He presented the case of a negro with a lesion which clinically was diagnosed granuloma pudenda, but pathologically, squamous cell epithelioma. The patient was shown again after treatment with antimony potassium tartrate and he was entirely cured. Carcinoma diagnosed by the pathologist is typical enough for him to make that diagnosis; it is not comparable to the borderline cases of inflammatory diseases or the granulomas in which there may be a difference of opinion. A diagnosis made by a reputable pathologist should bear some weight.

DR. SATENSTEIN: A positive microscopic diagnosis of carcinoma is seldom made when carcinoma is not present, and a microscopic diagnosis of nonmalignancy is often made when there is a carcinoma, the reasons being faulty interpretation and poor specimen. Misinterpretations are not uncommon. In a number of granulomas of long duration there is marked acanthosis and distortion of the follicle walls. When the tissue is sectioned at a slant by faulty technic, islands

of epidermal cells are noted in the deep cutis, which often resemble whorls; and when a hyperkeratotic follicle is cut at a slant, it resembles a pearl. If these areas of cells are studied serially, it is readily noted that they are part of an inflammatory process and not of a neoplasm. Again, what is more important than the islands of cells or misinterpreted pearls, is the character of the tissue reaction, and the presence or absence of anaplasia. These are more diagnostic than those just mentioned. I have already alluded to poor specimens in the discussion of the case of gumma of the leg presented by Dr. Cannon.

DR. CANNON: It is not unusual for granuloma inguinale to be mistaken for carcinoma both clinically and pathologically. You may recall the case I presented before this body over a year ago, sections from the lesions of which were diagnosed by three prominent pathologists as carcinoma. The ulcers in that case healed following injections of antimony potassium tartrate. I believe, with Dr. Pollitzer, that the general pathologist is not sufficiently familiar with pathologic processes of the skin to avoid incorrect diagnoses in such cases.

DR. POLLITZER: I recall the case of a young physician who had a small ulceration on the end of his index finger. Some one suggested that it looked like an epithelioma; a biopsy was made and the diagnosis of epithelioma was corroborated; the entire lesion was excised, and the wound healed. A few months later the man developed a roseola, and ultimately died of general paralysis. That is the sort of thing that excellent pathologists are doing all the time. Of course, this tissue was excised obliquely, and they mistook islands of cross-cut rete pegs for epithelioma.

BISMUTH ULCERATION OF TONSILS. Presented by DR. MCCAFFERTY.

A. P., a housemaid, aged 40, presented herself at the service of Dr. Dillingham in the Polyclinic Hospital, on May 16, 1923, with a syphilitic cirrhosis of the liver, and was at once placed on antisyphilitic treatment. She was given neoarsphenamine and three full courses of bismuth; during the last course pain and ulceration of the tonsils with pigmentation developed. All treatment was stopped, and the condition improved.

The Wassermann reaction was still four plus after three years of energetic treatment. The smear from the tonsils showed *staphylococcus albus,* numerous bacilli and an occasional spiral organism. Diphtheria bacilli were not found.

XERODERMA (?). Presented by DR. BECHET.

L. F., a boy, aged 7½ years, had had some scaly, dry lesions on the ears and neck for an indefinite time. On the neck they were rounded, sharply outlined and suggestive of tinea. Two microscopic examinations of scrapings were negative. There was a dry and scaly condition on the legs and arms, keratotic in character and resembling keratosis pilaris. On the back and sides of the neck were grayish, spiny, epidermic pegs, which had a tendency to group arrangement.

DISCUSSION

DR. WISE: From the diagnostic point of view, the case is interesting and decidedly puzzling. I examined the patient carefully and believe that the lichen pilaris lesions are independent of the scaly lesions on the neck and hair line. Any child of that age that does not bathe frequently is liable to have as much lichen pilaris as this child. It seems to me that a closer investigation should be made with regard to fungi in the scaling eruption.

MULTIPLE BENIGN EPITHELIOMA. Presented by DR. GOODMAN. (From the Skin and Cancer Hospital.)

M. N., a white woman, aged 21, said that the lesions on the skin first appeared when she was about 14, first on the left side of the nose near the mouth. The lesions had never given any subjective symptoms. No member of the family had ever had any similar lesions.

She was first seen at the Hospital on March 10, at which time she presented many lesions on the face varying in size from that of a pinhead to that of a pea. They appeared to have a predilection for the area about the eyes, the ala nasae and the upper lip. The back showed many lesions—most of them were small but a few were large, almost twice the size of a pea. Several lesions were excised and several were treated with electrodesiccation. All the wounds caused by excision healed; most of those treated with electrodesiccation had regressed, though some seemed as active as previously.

The microscopic appearance was that of a basal cell epithelioma. The nests of basal cells did not show any connection with the basal cell layer of the epidermis nor with the hair follicle. In fact, evidence of any connection of the nests with other structures was not seen in any of the sections examined. Cystic formation was not noted in the sections studied.

PEMPHIGUS FOLIACEOUS. Presented by Dr. Cannon.

M. D., an unmarried colored woman, aged 42, born in the United States, had a condition which began with blisters in May, 1923, followed by crusting and scaling over the scalp and neck, between the shoulders and on the face and which gradually spread over the body and extremities. The patient felt tired and weak most of the time. She was in Bellevue Hospital for two years, her condition being diagnosed as pemphigus.

Examination showed a moist, scaly condition of practically the entire cutaneous surface. The epidermis could be removed in large sheets, leaving a smooth, red, shiny surface; there were a few small ulcers in the mouth. General physical examination, a Wassermann test and blood count examination were negative. Treatment consisted of iron cacodylate administered intravenously, coagulin intramuscularly, alkalies, arsphenamine and mercurochrome-220 soluble. Improvement was most marked after the alkalies.

HODGKIN'S DISEASE. Presented by DR. SHARLIT.

This patient had first been shown and discussed at the December meeting of the section. Since then, a biopsy taken from the prepuce showed a granuloma.

PEMPHIGUS VULGARIS. Presented by DR. SPIEGEL.

C. S., an Austrian Jewess, had been operated on ten years previously for goiter; and one year prior to presentation had received two radium treatments for metrorrhagia, at the General Memorial Hospital. The eruption appeared on the nape of the neck four weeks prior to presentation.

Examination showed numerous discrete round, oval and irregularly shaped vesicles and bullae, varying in size from that of a pinhead to that of a pea and larger. Some of them were tense, others flaccid. The contents of the blebs were clear at first; later they became turbid. The individual lesion appeared to arise from the apparently healthy skin, an areola appearing at the base of the vesicle or bulla after a time. Many of the vesicles and blebs had become confluent, and

when ruptured formed yellow crusted and in some areas hemorrhagic lesions, the bases showing denuded areas. Nikolsky's sign was not present, but at the site of the Wassermann needle puncture a large vesicle appeared.

The Wassermann reaction was negative and the results of urinalysis were normal. The blood count was: neutrophils, 66 per cent; lymphocytes, 34 per cent.

Contents of vesicles showed neutrophils, 43 per cent; eosinophils, 42 per cent, and lymphocytes, 1 per cent. The patient weighed 99 pounds (44.9 Kg.). There had not been any loss of weight.

PEMPHIGUS VULGARIS. Presented by DR. TRAUB.

Mrs. R. I., aged 49, a Russian Jewess, presented a condition that began seven weeks previously as a bullous eruption confined to the buccal mucosa. She had no lesions on the surface of the skin, although she complained that at times her skin felt itchy. Directly before the onset of the trouble she felt pain, tenderness and a rather full sensation over the epigastrium. A few days before presentation bullae arising from the normal skin had appeared on the back.

Repeated smears for Vincent's angina had been negative. The Wassermann reaction was negative.

ERYTHEMA AB IGNE. Presented by DR. TRAUB.

Miss R. M., aged 19, born in New York, had an eruption on the legs that had been present for the past nine months. She gave a history of using a hot water bottle at the bottom of the bed at night as well as a heater near her legs while at work. It was a typical case.

MELANODERMA (?) Presented by DR. MCCAFFERTY. (From the Vanderbilt Clinic.)

J. S., a carpenter, born in the United States, said that most of his work was done in the open air, and that his sleeves were usually rolled up. In August, 1926, he started to use some composition containing creosote, and in September the skin condition began to appear. On the face and forearms was an erythematous and pigmented eruption of a mottled appearance. He also presented leukoplakia of the oral mucous membrane.

MYCOSIS FUNGOIDES. Presented by DR. MALONEY for DR. HOWARD FOX.

The patient, C. D., had been presented at the December, 1927, meeting. Treatment by the roentgen rays for the past three months resulted in considerable improvement.

DERMATITIS MEDICAMENTOSA (PHENOLPHTHALEIN). Presented by DR. MALONEY for DR. HOWARD FOX.

L. W., a man, aged 21, born in the United States, gave a history of having taken three days previously, three "boals rolls,' a phenolphthalein laxative. which was followed by itching of the penis and the appearance of several papules on the glans. A day or two later this was followed by similar lesions on the forearm. When first seen, he presented a violaceous patch on the flexor surface of the right forearm the size of a half dollar, and similar patches the size of a dime, well defined and circumscribed, on the left forearm, some involuting and just showing brownish pigmentation. On the penis were excoriations and exfoliations, probably the result of broken bullae. The palate showed broken bullae. This eruption had been recurrent for the past four years.

LICHEN PLANUS. Presented by DR. MALONEY for DR. HOWARD FOX.

P. B., a man, aged 71, born in the United States, gave a history of having had an eruption nine years previously, with occasional exacerbations. On the flexor surface of both wrists he showed a violaceous, purpuric eruption consisting of papules, some telangiectasia, and brownish pigmentation. On the shaft of the penis was a group of scratched papules. Over the tibia and instep was an eruption consisting of telangiectasia and purpuric patches, closely aggregated. On the right buccal membrane were a few white patches the size of a pea, while on the left side were large, white areas.

PERFORATING ULCER OF THE HEEL. Presented by DR. CANNON.

G. F., a white man, aged 40, was admitted to the City Hospital on April 23, complaining of ulceration beneath the right heel, of ten years' duration, which began at the site of pressure from a shoe. He had frequent griplike pains in the ulcer. The lesion had not changed in character for several years. He had intermittent shooting pains in the legs. He gave a history of an initial lesion and gonorrhea ten years prior to presentation. He said that he had not had any secondary lesions.

Examination showed a deep ulceration beneath the right heel, extending to and exposing the tendon. The margins were irregular and verrucous. At one part of the margin was a pigmented area, resembling an old blood clot.

The patient had Argyll-Robertson pupils, absence of deep reflexes and a positive Rhomberg sign. The blood and the spinal fluid Wassermann reactions were positive.

ACRODERMATITIS CHRONICA ATROPHICANS. Presented by DR. CANNON.

M. K., a man, aged 62, was admitted to the City Hospital March 3, 1927, complaining of a red, wrinkled condition of the skin over the extremities, of about twenty-five years' duration. The condition began on the back of the fingers and hands and back of the feet, with a red, puffiness of the skin. He had had frequent attacks of arthritis in the joints. He had had typhoid fever twenty-five years before, and erysipelas twenty years previously. He had always used alcohol in excess.

Examinations showed a smooth, soft, atrophic, cigaret paper-like condition of the skin, with the veins shining through, most marked on the extensor surfaces, and extending up on the hips and lumbar regions, where there was a marked demarcation to the lesions. On the outer surface of the left forearm was a linear, bandlike thickening about 8 inches by 1 inch (20.3 by 2.5 cm.) in diameter. A general physical examination showed arthritis of many of the joints; a systolic murmur at the apex and base of the heart. The blood pressure was 180 systolic and 90 diastolic; a Wassermann test and examinations of the urine gave negative results; the chemical examination of the blood was normal.

EXTENSIVE PSORIASIS OCCURRING IN A MAN AGED 65. Presented by DR. CANNON.

J. B., aged 77, was admitted to the City Hospital on March 15, complaining of a generalized eruption which had recurred at intervals for ten years. The hands had been so extremely involved as to render them almost useless.

On the hands and fingers were scaly, crusted, sharply circumscribed plaques covering an erythematous skin. The nails were greatly thickened and grayish yellow, with marked keratosis beneath them. On the body, extremities and scalp, were large typical psoriatic plaques.

PARAFFINOMA. Presented by DR. THORNLEY and DR. ABRAMOWITZ. (From the Gouverneur Hospital.)

Mrs. R. G., a Russian woman, had been in the United States eight years. She had had three children and there had not been any miscarriages. She gave a history of typhoid fever eight years previously, when she received a number of hypodermic injections. Ever since then she had had a small tumor on the back of the left arm. For the past month this growth had increased in size. It was not well defined, and was about the size of a small orange. It was slightly painful and gave an indistinct feeling of fluctuation at the center, and the surface temperature was a little elevated. There was also a linear scar at about the same location on the right arm.

TUBERCULOSIS COLLIQUATIVA. Presented by DR. MCCAFFERTY. (From the Vanderbilt Clinic.)

F. A., aged 4, born in the United States, presented a lesion on the skin, the duration of which was unknown to his mother. On the right side of the cheek were two coin-sized, elevated, red lesions of rather soft consistency. There was some seropurulent thin discharge from a sinus in each of the lesions. On the left hip was a slightly pigmented and scaly patch. Bacteriologic examination was negative.

PEMPHIGUS VULGARIS. Presented by DR. CANNON.

Mrs. M. G., aged 60, a Russian housewife, was admitted to the City Hospital five months prior to presentation complaining of recurring blisters over the entire cutaneous surface, with the exception of the face. The condition had begun eleven months earlier, with blisters on the scalp and on the backs of the hands, followed a few months later by lesions over the entire body and in the mouth.

Examinations showed a general bullous eruption. The bullae varied in size from that of a pinhead to that of a palm, and occurred on any otherwise normally appearing skin. They were observed especially over the abdomen, back and buttocks, where large areas were denuded of epithelium, leaving a raw, granular bleeding surface. General physical examination and the Wassermann reaction were entirely negative. The urine showed only a faint trace of albumin. Cultures of the blood and bullous fluid were negative. The chemical examination of the blood showed: urea, 14 mg.; sugar, 18 mg. Treatment consisting of alkaloids, followed by small doses of neoarsphenamine, resulted in pronounced improvement.

PSORIASIS AND LICHEN PLANUS. Presented by DR. BECHET.

C. B., a woman, aged 25, stated that she first noticed scaly plaques on her arms and legs ten years previously. She had an extensive psoriasis, several of the patches being several inches in diameter. The eruption seemed particularly bad on the arms. There were also some pigmented resolving lichen planus lesions on the arms, and here and there was seen the typical polygonal, violaceous, umbilicated lichen planus papule.

LYMPHANGIOMA. Presented by DR. MALONEY for DR. HOWARD FOX.

L. O., aged 13, born in the United States, had had an eruption on the right hip since infancy. She had been suffering with tuberculous arthritis of the hip joint for many years and had been operated on for this twice. On the right hip, extending from the anterior surface of the leg to the gluteal region was a large, deeply pigmented, verrucous growth, consisting of closely aggregated, glistening papules, the size of a pinhead, opalescent vesicles and numerous soft nodules which were easily compressible.

FOX-FORDYCE DISEASE. Presented by DR. MALONEY for DR. HOWARD FOX.

M. S., a housewife, aged 26, born in the United States, presented in both axillae a palm-sized area consisting of closely aggregated, sharply defined, infiltrated, hard, discrete papules varying in size from that of a pinhead to that of a pea, a good many of them being pierced by hairs. The itching was intense. This condition had been present for seven years. Biopsy disclosed chronic inflammation.

DERMATITIS HERPETIFORMIS (?). Presented by DR. MALONEY for DR. HOWARD FOX.

M. B., single, white, aged 18, had noticed two patches above both shoulder blades, about three months previously, and later, a slight eruption appeared on the left arm. The eruption consisted of groups of shiny papules, varying in size from that of a pinhead to slightly larger; some were slightly umbilicated and discrete. On the arm was an annular patch consisting of similar papules. Biopsy showed a pathologic picture of dermatitis herpetiformis.

RUPIAL SYPHILID. Presented by DR. MALONEY for DR. HOWARD FOX.

L. H., a colored woman, aged 43, born in the United States, presented a thickly crusted, rupial eruption on the nose extending from the base to the tip, and on the right side of the upper lip. It had begun two years previously as a small "sore" on the upper lip. A Wassermann test gave a four plus reaction. There was not any history of primary or secondary syphilis.

SECONDARY SYPHILID WITH NEUROSYPHILIS. Presented by Dr. CANNON.

Mrs. F. R., a widow, aged 45, was admitted to the City Hospital, April 1, 1927, with a generalized maculopapular, nonpruritic eruption of three weeks' duration. She said that she had not had initial lesions. The past history was negative.

The patient presented a generalized maculopapular eruption, most marked on the trunk and face. The lesions were the size of a pea and brownish red, with a tendency to grouping. There was a generalized adenopathy, most marked in the inguinal region, also a vaginal ulcer and gonorrhea. The patient also showed Argyll-Robertson pupils, reflexes of the left arm more active than those of the right; the left knee jerk more active than the right; and an absence of the ankle jerks. The blood Wassermann test was four plus.

RECURRENT ROSEOLA. Presented by DR. PAROUNAGIAN and DR. MASON.

B. E., aged 30, was seen at the Bellevue Clinic on Nov. 1, 1926, with a roseola, a scar of a chancre at the sulcus, of three months' duration and a Wassermann reaction of four plus. He was given two injections of silver arsphenamine and two of mercuric salicylate during November, 1926. He returned in January, 1927, and received two more injections of mercury; then he remained away until March 29, 1927. At this time, he showed a faint macular eruption which was similar to ordinary roseola, with the exception that a number of circinate lesions were noted, which were characteristic of recurrent roseola. In addition he had a number of moist papules at the sulcus where the original chancre occurred. The duration of the existing roseola was one week, and of the sulcus lesions two weeks. The Wassermann test was reported four plus.

CHANCRE OF TONGUE. Presented by DR. PAROUNAGIAN and DR. MASON.

M. N., aged 34, referred to the Bellevue Syphilis Clinic on March 29, 1927, presented an eroded chancre on the dorsum of the tongue, to the right of the median line. It was circular and about the size of a dime. The patient thought

that he had had the condition about eight weeks. He showed a profuse macular eruption, and the submaxillary glands were swollen on both sides. The Wassermann reaction was four plus.

CHANCRE OF LIP. Presented by DR. PAROUNAGIAN and DR. MASON.

F. D., aged 26, came to the Bellevue Syphilis Clinic on March 29, 1927, presenting a large, indurated lesion on the lower lip, which he said was of five weeks' duration. He had a generalized roseola and marked swelling of the submaxillary glands on both sides. The Wassermann test was four plus.

NODULAR CIRCINATE SYPHILODERM OF THE FACE AND SCALP. Presented by DR. PAROUNAGIAN and DR. MASON.

J. G., aged 50, an Italian woman, was admitted to the clinic on March 30, 1927, with an eruption which first appeared in front of the left ear as a small papule, and gradually extended to the other parts of the face and scalp. It consisted of thick, crusted, ulcerating and circinate patches with elevated borders; the centers were erythematous and dark brownish, involving the flush area, forehead, left ear and frontal portion of the scalp. The condition closely resembled lupus vulgaris; in fact, that diagnosis had been made by some physicians. The right ear and region around it were not affected. The Wassermann reaction was four plus.

GEORGE C. ANDREWS, M.D., *Secretary.*

MINNESOTA DERMATOLOGICAL SOCIETY

Regular Meeting, April 6, 1927

H. E. MICHELSON, M.D., *President*

LUPUS VULGARIS (FIVE CASES). Presented by DR. FREEMAN.

DISCUSSION

DR. FREEMAN: I do not think there is any question about the diagnosis in these cases; the question is one of treatment.

DR. SWEITZER: In the case of the elderly woman, the patient is probably a little bit sensitive about the scar, but I do not believe one can treat the patient without causing scars. Personally, I would prefer in that type of case to take acid nitrate of mercury and go over a small area at a time. It looks to me as though she had an enlarged gland on one side. I would treat that with the roentgen ray. In cases of infection of the mucous membrane acid nitrate gives good results. If it is possible to give treatments with the arc light as in some of the institutions in Europe, I imagine that would help.

PAPULONECROTIC TUBERCULID AND ERYTHEMA INDURATUM. Presented by DR. MICHELSON.

A woman, aged 24, said that the present eruption had been present several months, and that she had had similar eruptions previously which had appeared in crops and then disappeared without any particular treatment. At the time of presentation there were several deep-seated, globular masses to be palpated, situated in the hypoderm at the posterior region of the lower part of the legs.

The skin overlying these nodes was somewhat scaly and a deep purple. In certain locations the skin was depressed and adherent to the underlying nodule. On the backs of the hands and on the shell of the ear were several small, hard, shotty papules which showed a distinctly necrotic center. The patient had been examined for systemic tuberculosis, and a negative report had been received.

ERYTHEMA NODOSUM (UNILATERAL) WITH ERYTHEMA MULTIFORME-LIKE LESIONS. Presented by DR. O'LEARY and DR. MONTGOMERY.

Miss M. S., aged 21, had been a patient at the Mayo Clinic on various occasions since 1922. When about 2 years of age, she had had an attack of infantile paralysis which had affected the right leg. An astragolectomy was done in September, 1922. There were no lesions of the skin prior to the operation. Since then she had had recurrent lesions limited to the right leg; these lesions disappeared during the summer. She also had a fused kidney and a basal metabolic rate of minus 10, acne vulgaris and probably anorexia nervosa. The patient was seen on Jan. 27, 1927. A diagnosis was made of erythema multiforme because of the iris-like lesions which she presented. Further observation made us believe it was a case of erythema nodosum, at times accompanied by erythema multiforme-like lesions. Biopsy showed features of both diseases.

TUBERCULOUS CERVICAL ADENITIS, ERYTHEMA INDURATUM AND LICHEN SCROFULOSORUM. Presented by DR. MICHELSON.

A woman, aged 26, said that for the past few months she had felt as well as usual, and, although she had not lost decidedly in weight, she was suffering from chronic fatigue. Recently she had noted that her feet were somewhat swollen at night, and that an eruption had appeared over the body and at the calves of the legs. On examination it was noted that the posterior cervical glands were enlarged, some of them to the size of a walnut, and that over the trunk, especially on the anterior abdominal wall, there was a diffuse eruption consisting of sharply outlined, minute, yellowish, scaly papules. No particular arrangement was noted, but in certain areas the papules had coalesced into fairly large plaques. In the region of the calf there were cyanotic colored, scaling plaques which were immediately over deep-seated nodes. There was no ulceration.

DISCUSSION ON THE CASES OF DRS. MICHELSON, O'LEARY AND MONTGOMERY

DR. MICHELSON: I want to confine my discussion to the last case. This woman was carefully examined by a specialist in tuberculosis and sent to me with a diagnosis of varicose eczema. I think that we should teach medical students more about tuberculosis of the skin. It is illuminating to me that in the training of tuberculosis specialists little time is devoted to the cutaneous aspects of the disease.

DR. ARMSTRONG: I would like to ask about tuberculin. I know it has gone out of style somewhat, but I find that some of these patients improve considerably with the administration of $\frac{1}{100.000}$ mg. of tuberculin, tuberculin residue (new tuberculin).

DR. GAGER: I use tuberculin intradermally in $\frac{1}{100.000}$ dilution, and all my patients will not tolerate more than that. I have tried increasing the dose to $\frac{1}{50.000}$ but there is a severe reaction in all of my cases. I have treated two patients with the roentgen ray, but I do not thing much of the results.

DR. ARMSTRONG: In this treatment a reaction is not desired, as this would make the patient's condition worse.

DR. TURNACLIFF: Along the line of treatment with tuberculin, I might mention that recently I had a man with furunculosis on the back of his neck. He also had tuberculids of long duration. I gave him turpentine in oil, and it certainly helped the tuberculids. That may be along the line of the reaction to foreign protein.

DR. MONTGOMERY: I should appreciate any therapeutic suggestions regarding the patient with the unilateral erythema nodosum. We have given her autogenous vaccines, with some improvement. One may perhaps attribute the unilateral distribution to vascular disturbances due to an old infantile paralysis.

DR. OLSON: The association of erythema multiforme and erythema nodosum is not uncommon. In his textbook, MacLeod includes erythema nodosum under, or as a variety of, erythema multiforme. Salicylates might be beneficial to this patient.

LUPUS ERYTHEMATOSUS (TWO CASES). Presented by DR. FREEMAN.

LUPUS ERYTHEMATOSUS. Presented by DR. SWEITZER.

A man, aged 31, first noticed lesions in the summer of 1926. On Feb. 19, 1927, we began to administer 100 mg. of gold sodium thiosulphate intravenously every week. To date he has received five doses. He has also received 1/15 grain of gold chloride (0.00432 Gm.) three times a day for three weeks. He had a local reaction after the last intravenous treatment one week ago, which has subsided. At present the patient is much improved.

DISCUSSION ON THE PAPERS OF DRS. FREEMAN AND SWEITZER

DR. MONTGOMERY: In regard to 4 amino-2 auro thiophenol carbonic acid, Drs. O'Leary and Goeckerman have used it in twelve cases, with definite improvement in four of them. They have avoided using it in the disseminated cases because of a tendency to dissemination as noted in one patient with the discoid type of the disease following three injections.

DR. MICHELSON: Are you favorably impressed with the gold treatment?

DR. MONTGOMERY: We have had a definite improvement in one third of the patients treated with amino auro thiophenol carbonic acid.

DR. SWEITZER: We have just a small series of cases, and I have treated one private patient with gold and sodium thiosulphate. At our last meeting I presented one patient with a lesion on the side of the chin. He did not have any reaction. The skin is dark, but I do not know of any way to improve that. The other three of our patients have all had local reactions. The patient I presented tonight had been given five injections. In checking up Schamberg's work on the use of this gold treatment, I find that he recommended an initial dose of 50 mg. Reports from the Research Laboratory of Philadephia stated that the dose was 100 mg. once a week. I have followed that line of treatment, and have given it in 2 cc. of distilled water intravenously. I have not had a large enough series to know whether or not this will be effective. Schamberg speaks of the local reaction, and claims that he often obtains a local reaction and that the patients are benefited.

DR. MICHELSON: We have used the gold treatment for lupus erythematosus in only two cases, but we are impressed with the results so far obtained. It is interesting to note that constitutional therapy can produce a local effect in

lupus erythematosus and confirms the view that lupus erythematosus is a local manifestation of a constitutional condition.

DR. BOREEN: In regard to the case of lupus erythematosus in which there was atrophy of the hands, it seems to me there is idiopathic atrophy of the skin.

DR. MONTGOMERY: It seemed to me that there were atrophic lesions on the wrists, but there were definite erythematous lesions on the backs of the hands that would suggest a dissemination of the lupus erythematosus.

BLASTOMYCOSIS. Presented by DR. FREEMAN.

LESION ON FOREHEAD. Presented by DR. FREEMAN.

DISCUSSION

DR. MICHELSON: Because the great majority of ulcerations of the head are epitheliomatous, one is prone to make such a diagnosis quickly, but this case of Dr. Freeman's brings out the fact that every ulcer of the head is not a cancer and that syphilis must constantly be kept in mind.

PEMPHIGUS. Presented by DR. O'LEARY and DR. MONTGOMERY.

Mr. L. W., aged 20, developed intermittent fever in August, 1925, after swimming in Lake Michigan. In November, 1925, he had an attack of generalized urticaria. In December, small vesicles appeared in the axillae, followed by small and larger vesicles and bullae on the trunk and extremities. When the patient was seen at the Mayo Clinic, on Feb. 8, 1927, he presented an ocular palsy and a left vasomotor paralysis in addition to the bullous lesions. This was of only ten days' duration and cleared up after that time. The general examination was essentially negative, except for septic tonsils which were removed, and a prostatitis. A culture of the spinal fluid was negative. The patient has shown definite improvement under the Davis' treatment and treatment for prostatitis.

DISCUSSION

DR. MONTGOMERY: This case of pemphigus is interesting as the man has atrophic changes of the palms of the hands suggesting a dysgenetic background. At times, also, he presents a picture of erythema multiforme rather than one of pemphigus. He is presented as having a case of pemphigus.

DR. SWEITZER: I had a patient in the General Hospital a year or so ago who looked as though he had erythema multiforme on the hands and feet and a bullous eruption over the entire body. I made a diagnosis of pemphigus. Dr. Michelson thought it was erythema multiforme. I gave him the Davis' treatment, and he became worse. I thought he was going to die. I finally gave him salicylates in heavy doses; the lesions gradually healed, and the patient now feels well.

DR. MICHELSON: Did you doubt the diagnosis of pemphigus in the case shown?

DR. SWEITZER: Not necessarily, but the patient has so many other symptoms. One lesion on the arm looked like typical pemphigus. I merely made the suggestion that he might have some other condition also.

DR. MONTGOMERY: This patient has improved under the Davis' treatment. Prostatic massage has also been given because of a highly infected prostate.

DERMATITIS ON THE HANDS. Presented by DR. FREEMAN.

DISCUSSION

DR. BOREEN: This seems to be a seasonal condition.

DR. FREEMAN: This is a seasonal condition and clears up in the summer.

DR. SWEITZER: I think there is a large group of cases of this type. I look on these as sensitization cases. Some patients develop the condition seasonally and later have it all year. They are about the most difficult cases to do anything with. Lately I have been prescribing continuous bath, and I have had better results with this than with any other treatment.

LICHEN PLANUS. Presented by DR. BOREEN.

Miss J., aged 28, a University Dispensary patient, presented numerous closely segregated, violet papules over the sternum. The mucous membranes and the rest of the body were free.

LICHEN PLANUS. Presented by DR. TURNACLIFF.

A man, aged 32, presented an eruption on his wrists, on the sides of the body and on the glans penis, which he had had for three years. The lesions were papules with flat tops, coalescing to form plaques on his wrists and body and a definite circle on the glans penis.

DISCUSSION ON THE PAPERS OF DRS. BOREEN AND TURNACLIFF

DR. GAGER: The last case of lichen planus I had was that of a woman, aged 50, whom I saw in March. She had typical lichen planus, and I gave her proto-iodide pills, and in less than a month the lichen planus had disappeared. Another patient I recall having seen several years ago had lichen planus on the penis. He had a primary lesion and was under treatment. At the end of a year, he developed lichen planus, and I had to keep on treating him. I did not want to give him arsphenamine; so I gave him injections of mercury. It required six months of mild mercurial injections before the lichen planus on the penis cleared up.

POIKILODERMA OR GENERALIZED LICHEN PLANUS ERYTHEMATOSUS. Presented by DR. MICHELSON.

A woman, aged 43, said that for the past two years she had had a more or less generalized eruption over her trunk and extremities, which itched considerably. During that time her general health had been undermined, she suffered much with gastric trouble and had lost 35 pounds (15.9 Kg.) in weight. General examination, which was most thorough and complete, did not reveal any organic disease. Cutaneous symptoms were as follows: There was a generalized eruption over the trunk and the lower extremities. This consisted of a diffuse, brownish and purple eruption which appeared to be in the skin rather than on the skin. In areas the integument was distinctly atrophic and was no thicker than thin tissue paper. In other areas, there was marked telangiectasia, and in other areas, minute angular, deep-seated, reddish-purple papules were noted. The mucous membranes were normal.

DISCUSSION

DR. SWEITZER: Is it possible that she has an infection of the gallbladder? This sort of case looks as though the patient had some infection that has not been found.

DR. MICHELSON: Disease of the gallbladder was ruled out by the roentgenograms.

DR. PAGE: There is nothing that I can add. The condition is what I had considered chronic osteitis; the patient had periodic attacks of vomiting, and I sent her to the University Hospital.

DR. OLSON: I am inclined to favor the diagnosis of lichen planus in spite of the lack of infiltration in the microscopic sections. In the declining stage of lichen planus there may be little or no infiltration.

DR. MONTGOMERY: I have never seen a case of poikiloderma, but the condition resembles acute lichen planus in the stage of involution in which it is difficult to find the characteristic papules.

DR. MICHELSON: We have not arrived at a definite diagnosis in this case. The microscopic picture was not pathognomonic of any particular condition. We are going to treat the patient for lichen planus and watch the result.

MORPHEA-LIKE EPITHELIOMA. Presented by DR. ARMSTRONG.

A woman, aged 52, had a lesion on the left cheek eleven years before presentation; it has now reached a size of 1 by 1½ inches (2.5 by 3.97 cm.). It has healed, with the exception of three small areas on the upper edge which were frozen with carbon dioxide snow three weeks ago. These areas now appear to be healed.

BASAL CELL EPITHELIOMA OF THE MORPHEA TYPE. Presented by DR. MICHELSON.

A man, aged 32, because he had pulmonary tuberculosis, had had the lesion on his face diagnosed as lupus vulgaris. On examination, a lesion on the left cheek was noted which was about 2 inches (5 cm.) in diameter, of circular outline, with a well defined, rolled, translucent border and small vessels growing over the border. The center of the plaque was ivory colored and contained nodules appearing much the same as the border. Biopsy showed basal cell epithelioma. Roentgen-ray therapy was to be given.

PIGMENTATION OF THE FACE AND NECK. Presented by DR. GAGER.

Mrs. O., a housewife, aged 54, presented a grayish pigmentation of the neck which encircled it like a collar and faded into the normal skin of the upper part of the chest. There was the same pigmentation at the angles of the mouth, extending downward onto the chin, and also at the angles of the nose and cheek. The pigmentation was darker in the nasolabial folds and in the lines at the angles of the mouth. The discoloration was symmetrical and presented a rather striking picture. The neck appeared unwashed, as Dr. Goeckerman first described this condition.

This pigmentation had been present for five or six years. The patient was a plethoric woman who had hypertension and symptoms pointing to a chronic infection of the gallbladder, although the roentgenogram did not show the presence of stone. Inquiry elicited the statement that the patient had habitually been using a cream which is known to contain a mercurial salt. The case was one of discoloration or pigmentation of the skin due to the use of a cosmetic.

DISCUSSION

DR. GAGER: I should like to ask if there is any treatment for the pigmentation.

DR. MONTGOMERY: There is no specific treatment for the removal of pigmentation other than stopping the use of the cream. In the course of a year

or a year and a half, the pigmentation decreases. Keratolytics have hastened this somewhat.

LESIONS ON THE TONGUE. Presented by DR. FREEMAN.

DISCUSSION

DR. FREEMAN: The patient has artificial teeth in both the upper and the lower jaw.

DR. SWEITZER: The first thing I would consider is pernicious anemia and the second is the artificial plates. Those made with rubber are the kind that have mercury in them. If these patients do not have anemia, I usually ask them to have a plate made without the rubber, metal being used.

DR. FREEMAN: It impressed me that the redness was not inflammatory; there is something the matter with the patient's tongue besides the condition caused by the plates.

RHINOSCLEROMA. Presented by DR. O'LEARY and DR. MONTGOMERY.

Mr. M. S., aged 26, was seen because of lesions in the nose, larynx and pharynx, of eight years' duration. He had always lived in this country. Hoarseness developed some years previously. Examination revealed extensive destruction of the nose, pharynx and larynx, including ulcerated granuloma of the alae. The patient had been under treatment at the Mayo Clinic for the past month, during which time a tracheotomy has been performed and radium treatments given. Biopsy for the left ala showed a typical histologic picture of rhinoscleroma, and bacilli were demonstrated. Cultures were positive for rhinoscleroma.

DISCUSSION

DR. MICHELSON: I have recently read Kaposi's description of rhinoscleroma, and note that he stated that he had seen about one hundred and fifty cases. He particularly brings out the fact that the lesions do not ulcerate. In this case presented by Dr. Montgomery, leprosy should be excluded.

DR. MONTGOMERY: Dr. Gans, in his textbook, says that histologically the lesions do ulcerate. The biopsy from the larynx shows the same histologic picture as that shown in the sections taken from the nostril. The condition is relatively common in central Europe, but is rare in this country. This case is of unusual interest in that, although the patient is of foreign parentage, he was born in this country and has never been out of the United States.

A CASE FOR DIAGNOSIS (CHILBLAIN PERNIO). Presented by DR. MICHELSON.

A woman, aged 18, presented herself because her hands and feet were constantly cold and of a bluish tint, especially throughout the winter. She said that her general health was good, and that, aside from the fact that the extremities became painful when exposed to cold for any length of time, she did not suffer any great inconvenience from her condition. General examination did not reveal a pathologic condition.

CHRONIC ROENTGEN-RAY DERMATITIS. Presented by DR. MICHELSON.

A woman, aged 25, said that three years previously she had received roentgen-ray treatments once a week from a physician in New York over a period of one year. Her face, on presentation, showed marked atrophy, telan-

gectasia and scarring. There were no ulcerations, but her face was dry, and because of the appearance was a decided detriment to her economic progress.

SEBORRHEIC DERMATITIS. Presented by DR. BOREEN.

R. S., aged 20, a University student, had a sharply marginated erythematous patch in the hairy portion of the pubic region and extending onto the shaft of the penis. A similar condition was found in both axillae, and there were several lesions on the scalp.

HEMORRHAGES UNDER THE SKIN. Presented by DR. SWEITZER.

George H., aged 74, white, single, who sold papers, said that two weeks before presentation a lesion had appeared on the right thumb. At first it was dark brown and then black. There was no history of injury. The lesion did not cause pain and did not become any larger. All the finger-tips of the right hand felt numb, as did also the lesion on the thumb. The left fingers were normal. The blood pressure was 180 systolic and 85 diastolic. The eye-grounds were not tortuous, and the disk was hazy. The results of general examination were negative. All of the reflexes were hyperactive. The heart was normal. The urine showed a few hyaline casts and a faint trace of albumin. Examination of the blood revealed: hemoglobin, 75 per cent; red blood cells, 3,850,000, and white blood cells, 8,700.

DR. HAMILTON MONTGOMERY (Rochester) gave a microscopic (lantern) demonstration and talk on rhinoscleroma and erythema induratum.

SCROLL-LIKE LESIONS ON CHEST. Presented by DR. BOREEN.

This was a further report on a case presented Feb. 2, 1927.

There was a difference of opinion when this case was presented at the February meeting. Most of the members of the Society thought that it was syphilis. The Wassermann reaction was negative. I first treated the patient with Whitfield ointment. The last time I saw her, more than a month ago, the lesion had disappeared. She is confined to bed with high blood pressure; so I have not seen her since. I think the diagnosis will have to stand as that of a fungus infection.

D. D. TURNACLIFF, Secretary.

MANHATTAN DERMATOLOGICAL SOCIETY

April 12, 1927

JOSEPH J. ELLER, M.D., *Chairman*

PEMPHIGUS VULGARIS. Presented by DR. WISE.

Mrs. M. B., white, aged 41, a Jewish housewife, said that in June, 1926, she noticed some blisters on the lower surface of the tongue, which looked like canker sores. These disappeared, but were followed by others on the tongue and on the inner sides of the cheeks. Shortly afterward she noticed bullae on her chest, abdomen, back and buttocks. During the next six months she used "all forms" of local applications without benefit.

On Dec. 6, 1926, she presented numerous vesicles and eroded superficial ulcers of the tongue and buccal mucosae. There were numerous blebs on her chest, abdomen and buttocks, and bullae arising from normal skin. Crusted lesions were scattered over the same areas. She had a foul-smelling breath and had lost 10 pounds (4.5 Kg.) in three months. She was treated with arsphenamine injected deep into the muscles of the buttocks every second day for two weeks; then it was given every day. The condition began to improve after the first injection. She also received roentgen-ray treatment, one quarter of an erythema dose to the chest and back, at first at seven day intervals, and later at fourteen day intervals. For six weeks the patient had not noticed a new lesion in her mouth and there had only been an occasional lesion on the skin of the back and the chest. The skin showed only pigmented areas. The patient was gaining weight and had resumed her household duties.

DISCUSSION

DR. MOUNT: I cannot make a diagnosis of pemphigus vulgaris at present with a duration of only seven months. However, I do not refuse the diagnosis.

DR. PAROUNAGIAN: I agree with the diagnosis of pemphigus vulgaris.

DR. SATENSTEIN: I do not doubt that the diagnosis is correct. For a number of years there have been a good many cases of pemphigus at the City Hospital, but I never saw a patient recover. Arsenic was administered to the saturation point, but the patients eventually died. I believe every one will admit that pemphigus vulgaris is entirely different from pemphigus foliacious and pemphigus vegetans. The interesting point in this case is that there has not been a recurrence of the lesions, but I believe that there will be. I think that the prognosis of pemphigus vulgaris is bad. I do not know of a single patient with true pemphigus vulgaris who has been cured.

The diagnostic possibilities in these cases should be considered further before calling the condition pemphigus from the clinical dermatologic observation only. The systemic manifestation often associated with this condition should be investigated. One is often confused in the diagnosis of pemphigus and other allied conditions. If the patient recovers from the condition, the diagnosis of pemphigus is often questioned.

DR. BECHET: In reference to Dr. Satenstein's remarks, I would like to call attention to a patient I saw some years ago. The clinical symptoms of pemphigus vulgaris were typical. There were lesions in the mouth, the bullae were large, Nikolsky's sign was present, the patient was weak and emaciated and the lesions had been present for two years. I gave an unfavorable prognosis. To my great surprise the patient began to improve under large doses of arsenic and eventually she recovered. To the best of my knowledge she has had no recurrences. Reports of cure from competent observers have been frequently noted. It is therefore possible to believe that true pemphigus, or a bullous disease which cannot be distinguished from it, occasionally does end in complete recovery. I am, therefore, willing to reject the dictum "that if the patient recovers it is not pemphigus."

DR. OULMANN: The case is interesting and I do not doubt the diagnosis. I will be interested to hear a report on the patient in the future. The only systemic change known to me is the retention of sodium chloride. The literature mentions a number of cases of pemphigus in which patients have been cured.

DR. WISE: The condition is pemphigus vulgaris. When I first saw the patient she had a foul-smelling breath and had lost weight. Those two signs

are characteristic of pemphigus vulgaris. She also had large bullae and no itching. The condition improved immediately after the administration of arsenic. I have had two patients with pemphigus vulgaris who have been cured. I think the secret of success lies in giving large doses of arsenic. The arsenic must be given in rapidly increasing doses, to the point of toleration.

LUPUS ERYTHEMATOSUS AND DERMATITIS MEDICAMENTOSA (GOLD). Presented by DR. WISE.

Mrs. A. B., aged 34, a housewife, was first seen on March 4, 1927, at which time she had had the eruption for one year. She presented a widespread, well-defined, bat-wing type of eruption affecting the nose, cheeks and extending down the sides of the chin and upper lip, and also affecting the back and sides of the scalp. In March, 25 mg. of gold and sodium thiosulphate was administered intravenously. She had eight injections in all, the largest being a dose of 100 mg. There was little improvement until the fifth injection, when the lesions became noticeably paler. After the sixth injection the patient had a constitutional reaction, which kept her in bed for two days with severe pains all over the body, a rise in temperature and a feeling of exhaustion. The condition improved markedly after the reaction. The seventh and eighth injections were followed by similar constitutional reactions and a striking improvement in the eruption. Three days after the eighth injection the patient developed a dermatitis. At this time all traces of the original eruption on the face, neck and scalp had disappeared, but the skin at the edges of the lesions was inflamed and edematous. The eruption resulting from the medication resembled that which occurs in erythema multiforme. It affected the skin of the face, neck, upper and lower extremities and the chest. It itched and burned moderately and gradually subsided under applications of calamine and zinc oxide lotion.

DISCUSSION

DR. OULMANN: I think the result here is good. This is the first case in which I have seen a skin reaction. I gave doses up to 200 mg. and did not see any reaction other than a slight malaise. I think it would be interesting to give this patient an injection of sodium thiosulphate to see if the eruption would clear up quicker.

DR. PAROUNAGIAN: The improvement in these cases is interesting.

DR. BECHET: I am interested in the reaction to the drug exhibited by this patient for the reason that I have now thirty patients with lupus erythematosus who have been treated with gold and sodium thiosulphate since last November. I use the gold and sodium thiosulphate advocated by Schamberg in doses of 100 mg. once or twice a week. There have been few reactions, certainly none so extensive as in the present case. Of the total number, I can recall but two cutaneous reactions and one systemic reaction. At times there have been local reactions consisting of burning and itching sensations in the lesions with an accentuation of color. Apparently the more active the local reaction the better effect the drug seems to have.

PERNIO. Presented by DR. WISE.

Miss L. R., aged 20, a bookkeeper, said that she first noticed the present condition about Christmas, 1926. She said that her hands were not exposed to cold before the eruption developed. The skin of the palmar surfaces of the fingers and thumbs was involved and to a less extent the backs of the fingers

at the distal joints and next to the nails. The lesions consisted of separate and confluent patches ranging from one quarter to 1 inch (0.62 to 2.5 cm.) in diameter, and were oval in shape and distinctly violaceous in color, and with a thickened and somewhat scaly surface. There was slight itching. The fingers and whole hand were swollen.

DISCUSSION

DR. BECHET: The condition is hard to classify, but it probably belongs to the erythema pernio group. If the patient had lupus erythematosus lesions on the cheeks one could not differentiate the lesions on the hands from disseminate lupus erythematosus. I do not consider the condition a true pernio, however, but rather place it in that ill-defined group closely related to the tuberculids.

DR. WEISS: I regard the condition as lupus pernio.

DR. PAROUNAGIAN: I agree with Dr. Wise.

DR. OULMANN: I regard the condition as belonging to the pernio group, owing to the acro-asphyxia of the hand and fingers, which I have never seen accompanying lupus erythematosus.

DR. SATENSTEIN: I do not know what diagnosis to make. The mere fact that no other portions of the skin are affected makes me hesitate to call the condition lupus erythematosus. There is not sufficient eruption of definite character to warrant the diagnosis of lupus pernio. I think that it will require further observation and study before a definite diagnosis can be made.

DR. WISE: True lupus pernio, as described by European dermatologists, is a granuloma. I doubt if this condition is lupus pernio. Lupus pernio is a productive disease with elevated edges, a granulomatous tumor, like sarcoid. This is a flat lesion. If accompanied by lesions on the face, the diagnosis, I am sure, would be lupus erythematosus. Owing to the fact that the patient has distinct acro-asphyxia of the backs of the hands, the diagnosis of pernio is applicable, but not lupus pernio.

LUPUS ERYTHEMATOSUS DISCOIDEUS TREATED WITH GOLD. Presented by DR. WISE.

G. E., a man, aged 46, white, a taxi starter, was first seen on Dec. 23, 1926, presenting an oval patch of chronic lupus erythematosus discoideus with an elevated, inflammatory, scaly border and a depressed, scarred and atrophic center. The patch extended from the region of the temple down the side of the cheek to the angle of the jaw. He had had the eruption for eighteen years, during which time he had received roentgen-ray, radium and solid carbon dioxide treatments without benefit. On December 23, he received his first injection of a gold compound, 25 mg. Since that time he had received twenty injections at intervals of from five to seven days. The dose was gradually increased to 100 mg. The improvement was gradual and the patient never had the slightest local or constitutional reaction. On presentation, the patch appeared to be cured; the only remnant of the lesion was an oval, smooth atrophic scar. The improvement became manifest after the fourth injection of the gold compound. Local treatment was not administered.

DERMATITIS MEDICAMENTOSA (PHENOLPHTHALEIN). Presented by DR. LEVIN.

A. F., a woman, aged 22, presented a phenolphthalein eruption with herpetic lesions in the mouth. The condition had existed from time to time for two years.

DISCUSSION

DR. SCHEER: Lesions in the mouth are not uncommon in cases of phenolphthalein eruptions. I presented such a case at the March meeting of the Section on Dermatology of the New York Academy of Medicine, and I know of several other cases in the practice of dermatologic colleagues.

DR. WISE: I have reported several cases in which there were lesions in the mouth and on the genitals.

EPITHELIOMA OF THE MOUTH. Presented by DR. LEVIN.

A man, aged 43, presented a painful, ulcerated condition of the mouth, of three months' duration. The left side of the tongue and roof of the mouth were involved by a large mass showing the presence of irregular growths and deep, excavated ulcerations, with marked adenopathy of the cervical nodes. The patient had received intensive roentgen-ray therapy to the glands on either side of the neck and back of the neck. The inflammatory reaction had greatly subsided since the administration of roentgen rays.

DR. WISE: Dr. Levin says that he intends to give this patient radium treatment. I approve, but I think the prognosis is extremely poor.

DR. MOUNT: I think the prognosis is extremely poor and that there will be only a temporary amelioration of the condition.

DR. OULMANN: I agree with the suggestion for radium treatment in this case and I think the prognosis is poor. When such a patient has intense pain, I have found that injections of colloidal selenium reduces the pain.

DERMATITIS HERPETIFORMIS. Presented by Dr. LEVIN.

A man, aged 76, complained of an intensely itchy, generalized eruption of ten months' duration. It first appeared as an itchy erythema on the trunk which gradually involved the entire body and then became bullous. He entered Mount Sinai Hospital two months previous to presentation and showed a condition resembling pemphigus. The lesions gradually faded leaving pigmentation and occasional bullous lesions. He had received various forms of treatment, including a nonspecific milk preparation, a lactalbumin preparation and autohemotherapy. Improvement occurred with autohemotherapy.

DISCUSSION

DR. BECHET: The therapeutic results are really remarkable, and if Dr. Levin believes that autohemotherapy alone is responsible, I would like for him to tell the quantity given at each dose, and the frequency of the injections.

DR. OCHS: This case is interesting to me, because when I saw the patient I sent him to Mt. Sinai with a diagnosis of erythema multiforme bullosum. He had had about four different physicians who had diagnosed his condition as pemphigus. He had a generalized eruption with bullous lesions in the mouth and on the scalp. There was intense itching. Wherever there were bullae he scratched them open. The greater part of the body was covered with crusts, but at the time I could not see any pigmentation. Now I see nothing but probable arsenical pigmentation.

DR. OULMANN: I agree with the diagnosis of Duhring's disease. The pigmentation is not due to arsenic but to the disease.

CONGENITAL SYPHILIS. Presented by DR. LEVIN.

A boy, aged 5, showed evidence of congenital syphilis with active lesions and a negative Wassermann reaction on the blood. The patient was admitted complaining of a painful swelling of the right leg of several months' duration. He showed a marked swelling over the front of the right tibia. Both tibiae were saber shaped, the teeth were of hutchinsonian type, the molars showed tubercles and the roentgenograms disclosed a syphilitic periostitis.

DIAGNOSIS—FOREIGN BODY TUMOR. Presented by DR. LEVIN.

H. F., a man, aged 49, had been infected with syphilis fifteen years previously, and had received injections of soluble mercury into the buttocks. From three to four weekly injections were administered for about three years. The year after the treatment was begun, the patient noticed swellings in the buttocks, which persisted. The patient showed evidences of general paralysis. Examination of the buttocks revealed tangerine-sized, bony, hard masses, not attached to the skin, but adherent to the deeper tissues. They were neither tender nor painful. The skin over the lesions was normal in color.

DISCUSSION

DR. SATENSTEIN: In the past dermatologists used to see a good many cases in which there were tumor masses in the gluteal regions. They were regarded as the result of faulty technic due to the deposition of medication in the fat, and they occurred most often when the needle was withdrawn. It is simply a reaction to the medication—a fibrosis. In two instances, chemical examination of a portion of the fibrosis following injections of mercury was negative to mercury. I would not regard these as foreign bodies in the sense of those noted following the introduction of paraffin and similar substances.

DR. LEVIN: This condition was described by Oppenheim. It is due to the injection of foreign bodies in the form of drugs like arsenic and insoluble mercury. It is not a paraffinoma which results from the fats used for suspension of the insoluble salts.

PALMAR SYPHILID. Presented by DR. PAROUNAGIAN.

Mrs. E. W., aged 26, had a large chancre on the upper lip, on Feb. 1, 1926, and reported to the clinic at Bellevue Hospital on March 29, 1926, with marked roseola and a four plus Wassermann reaction. After the administration of two injections of neoarsphenamine, one of arsphenamine and two of mercury, she developed jaundice which necessitated discontinuance of treatment for the time being. Later, seven injections of mercury salicylate, one of neoarsphenamine and one of silver arsphenamine were tried in minute doses, in the expectation of desensitization, without success; finally, twenty injections of bismuth were given from June 18 to September 3.

On September 15 and October 20, the patient's Wassermann reaction was negative. On December 29, she returned with a marked case of palmar roseola for which she was presented before the Manhattan Dermatological Society. The Wassermann reaction was again four plus. She was given twenty injections of bismuth from Jan. 14 to March 25, 1927. On March 30, the Wassermann reacion was plus-minus, and on April 3, she developed another palmar roseola. She had no other lesions on other parts of the body.

LUPUS ERYTHEMATOSUS TREATED WITH GOLD. Presented by DR. OULMANN.

The patient, aged 55, developed two lesions seven weeks prior to presentation, one at the tip of the nose and one at the neck in the region of the anterior auricular line. The lesions were round, varying in size from that of a dime to that of a five cent piece, with infiltrated margins and an adherent, scaly, keratotic center. The color was acute red. The patient had received two injections of a gold compound, and the lesions had almost disappeared.

BLASTOMYCOSIS. Presented by DR. OULMANN.

A butcher, aged 45, had an oblong lesion, 1 by 2½ inches (2.5 by 6.27 cm.) in size, on the bearded region of the chin. A diagnosis of sycosis vulgaris was made. The lesion was sharply circumscribed, the edges were raised and a crust covered the infected area. When this crust was lifted, the surface showed a verrucous condition with tiny sinuses containing pus. In the smear, as well as in a hanging drop, double contoured cells were found, and the budding mycelium of blastomycosis. Tincture of iodine and silver nitrate solution had been applied. There was no more crust formation, the verrucous condition smoothed down and only a few sinuses contained pus on presentation. The patient was to receive iodide internally.

DAVID L. SATENSTEIN, M.D.

BROOKLYN DERMATOLOGICAL SOCIETY

Regular Meeting, April 18, 1927

JOHN C. GRAHAM, M.D., *Presiding*

PURPURA ANNULARIS TELANGIECTODES. Presented by DR. THRONE.

J. W. A., a man, white, aged 20, showed an irregular, telangiectatic condition, small angioma, annular lesions, atrophy, and pigmentation on both ankles. The duration of the condition was several years. The atrophy had been present about two years. There was nothing important in the patient's previous history, except that he had "weak ankles." The family history was negative.

MYCOSIS FUNGOIDES. Presented by DR. GAUVAIN.

M. B., a woman, aged 42, married, had had an eruption since August, 1926, which began as a red spot the size of a quarter. The spot increased rapidly in size, and new ones developed for a period of four or five months. The condition had been diagnosed as "ringworm" and as "psoriasis." On March 15, at the Long Island College Hospital, the patient presented eczematous-looking patches, acutely inflamed, which improved under soothing applications. Pruritus was marked.

DISCUSSION

DR. SKEER: I think it is a case of the prefungoid stage of mycosis fungoides. The fact that the patient has the lesions on the face and hair line does not rule out premycosis.

DR. THRONE: I cannot see any psoriasis in this case. I believe that psoriasis does not show the distinct infiltration that this case does. I agree with the diagnosis.

KERATODERMA PALMARIS ET PLANTARIS. Presented by DR. GAUVAIN.

F. R., a boy, aged 11, had had a chronic, scaly, indurated lesion on the palms and soles for one and one-half years. He was presented before the Society in February, 1926 (*Arch. Dermat. & Syph.* **14**:206 [Aug.] 1926). He did not give any history of indigestion of arsenic, and was presented for suggestion as to treatment.

DISCUSSION

DR. GAUVAIN: In looking over the possible etiologic factors, I find that sometimes arsenic is the cause, but I gave the patient a therapeutic test of intravenous injections of sodium thiosulphate, reinforced by oral administration, without effect. He received a total of 3 Gm. in three weeks, and was getting 2 Gm. by mouth a day. Neither did he respond to the local use of salicylic acid in ammoniated mercury ointment.

DR. BERKOWITZ: I agree with the diagnosis. The boy also presents a marked ichthyotic condition of the legs, which I understand is associated with that disease.

DR. THRONE: I would continue the treatment by means of injections of sodium thiosulphate, giving 0.5 Gm. a week and 10 grains by mouth each day. I would use a 2 per cent salicylic acid ointment in a diachylon base locally. I have seen patients treated with the roentgen rays who did not respond, but I have never seen a patient who failed to respond to long continued use of sodium thiosulphate.

A CASE FOR DIAGNOSIS (PELLAGRA?). Presented by DR. WALZER.

C. F., a man, married, aged 24, a life resident of New York, had been exposed to venereal disease five months before presentation; six weeks later he had a lesion on the penis, but he never had any secondary symptoms of syphilis. He had also had "stomach trouble" for five years, which had been diagnosed as gastric ulcer. About four weeks before presentation, he had noticed his hands becoming red and sores forming on his lips. At presentation he had a pea-sized, infiltrated, healed ulcer on the corona of the penis. The tonsils had pea-sized, white ulcerations, and on the mucous membranes of the lips, particularly at the angles of the mouth, were pinhead-sized, white ulcerations. Near the mouth were two semi-annular lesions composed of papules and postules.

On the dorsum of each hand, involving the proximal halves of the fingers, there was a cyanotic patch, sharply demarcated and covered with adherent, fine scales. The plantar surfaces of the feet were similarly involved, but there was more keratosis.

DISCUSSION

DR. BERKOWITZ: I thought of pellagra as soon as I saw the patient. Of course, the diagnosis of syphilis is to be accepted, especially from the history, but pellagra is the condition to be considered in respect to the hands.

DR. SKEER: It is possible that the lesions on the hand are secondary syphilids. I think another diagnosis should be set forth, and that is some sort of infection —epidermophyton—of the hands. The border is pretty well marked and there are a few rather deep-seated vesicles. In addition, I believe that the patient has syphilis.

DR. THRONE: I think it would be advisable to wait for the results of the Wassermann test before syphilis is considered present. The cyanotic patches on the hands certainly look like pellagra.

NOTE.—The Wassermann reaction was negative. The patient developed all the classic symptoms of pellagra and died two weeks after presentation.

PEMPHIGUS VULGARIS. Presented by DR. PERSKY.

G. B. first noticed the condition about one and one-half years before presentation as small, circular, reddish patches situated over the chest, abdomen, thighs and legs. They soon spread over the forehead and scalp. These lesions commenced as an elevated patch slightly larger than a small pea, and soon became vesicular and crusted. The patient had received various forms of local and roentgen-ray treatment on the scalp and body. The last treatment was given about three months before.

Physical examination revealed a scalp almost completely epilated, with fine lanugo hairs here and there. The scalp was covered by a thick, tenacious, wax-like crust. On removal of the crust, bleeding points were seen.

Situated over the spine were approximately eight erythematous papules with a delicate covering, which could be removed easily. Directly below this area there was another similar grouping, except that several large flaccid blebs were seen arising from a normal integument and containing a thin, serous exudate. These erythematous patches were visible in groups over the chest and abdomen. The Nikolsky sign was present. There were no lesions of the mucous membranes.

DISCUSSION

DR. BERKOWITZ: I saw this woman about three or four months before presentation when she had a frank case of psoriasis and was treated for that. I have not seen her for about four weeks. At that time she did not have any lesions that suggested pemphigus. Now she shows some psoriatic lesions. She had the lesions on all the areas on which one would expect to find classic psoriasis.

DR. THRONE: Of course, one cannot tell now what the patient had months ago. From the present symptoms I would make the diagnosis of pemphigus. The Nikolsky sign is present and the lesions are typical.

DR. SKEER: I cannot see any evidence of psoriasis now. On the other hand, she presented several flaccid, irregularly-shaped, flat blebs, on an erythematous base. There was no itching.

LUPUS ERYTHEMATOSUS AND MYCOSIS FUNGOIDES. Presented by DR. THRONE.

The patient, L. W., was shown at the New York Dermatological Society, Nov. 16, 1926, by Dr. A. Schuyler Clark (*Arch. Dermat. & Syph.* **15**:225 [Feb.] 1927).

DISCUSSION

DR. THRONE: The combination of lupus erythematosus and mycosis fungoides is exceptional, as far as I know. I think that in 1892, Hallopeau presented a case before the French Dermatological Society, which was diagnosed by the members as mycosis. One year later, he showed the same patient, and the unanimous diagnosis at that time was lupus erythematosus. The patient presented here, came to the Skin and Cancer Hospital eight years ago with lupus erythematosus, for which she was treated with carbon dioxide snow and remedies of that nature. The result of this treatment is shown by the scarring. Five years ago she developed a general pruritus, and three and a half years ago the lesions began to develop on the body, trunk and forehead. Believing that this may be a case similar to that of Hallopeau, we have treated the patient

with a gold compound, thus far without beneficial results. The only changes that have taken place have been those due to mycosis fungoides. Some lesions have disappeared, but others have reappeared in other places. The gold injections may have caused the angry appearance of the facial lesions.

ARSENICAL POISONING. Presented by DR. THRONE.

I. D., a woman, aged 21, was referred to the New York Skin and Cancer Hospital Clinic on Dec. 1, 1926, from the neurological department of the Jewish Hospital of Brooklyn for treatment for motorocular paralysis of the left eye. She had eaten food which had been poisoned with arsenic. The summer before presentation, she had a severe nervous shock. The paralysis began about November 1. Results of the examination were negative except for paralysis of the third nerve. After injections of sodium thiosulphate, her urine showed arsenic in pathologic amounts. The patient had received injections of sodium thiosulphate, 0.5 Gm., three times a week, except for one week in January, when she showed a tendency to have vagotonia, and during that week she was given three injections of calcium chloride urea (Afenil). The paralysis was shown by ptosis of the lid and double vision. Recovery was complete.

E. ALMORE GAUVAIN, M.D., Secretary.

NEW YORK DERMATOLOGICAL SOCIETY

Regular Meeting, April 26, 1927

H. H. WHITEHOUSE, M.D., *President*

WHITE SPOT DISEASE. Presented by DR. LANE.

D. C., a boy, aged 4½ years, had an oval white lesion about 1 inch long by one-half inch wide (2.5 by 1.27 cm.), just external to the outer edge of the left eyebrow, which was said to have developed one and a half years previously, following a fall. There was no cut or bruise in this location at the time of the fall. The lesion had gradually increased in size. The center was somewhat hard and thick, and there was some exfoliation. The edges were smooth. The whole surface was white.

DISCUSSION

DR. MACKEE: The lesion, of course, is some form of scleroderma. I hesitate to designate it as white spot disease because of the absence of the peculiar infiltration that suggests a piece of exceedingly thin cardboard, (such as that used for playing-cards) embedded in the skin. However, in the late stages of white spot disease this infiltration may disappear and the milky white color may become less conspicuous. In white spot disease one does not encounter exfoliating hyperkeratosis, as is seen in the present case. In spite of the absence of a distinct violaceous areola, I am inclined to regard the lesion in Dr. Lane's patient as representing morphea.

DR. HIGHMAN: I agree substantially with Dr. MacKee. It is one of the primary atrophies, but to go into the differentiation between morphea and white spot disease would not be worth while.

DR. LANE: The child previously had some bald spots on the head, followed by a growth of white hair, undoubtedly alopecia areata. The hair on those spots has now changed to normal color.

DR. WISE: I agree with Dr. MacKee. According to Kyrle, the difference between white spot disease and morphea is that in white spot disease there is a degeneration of elastic tissue, while in morphea the elastic tissue is unharmed. As far as the diagnosis here is concerned, I do not think it easy; but I should say it was a patch of atrophying scleroderma, and that treatment with refrigeration would result in a cure.

DR. WHITEHOUSE: I agree with the diagnosis of morphea, clinically.

RINGWORM INFECTION OF ALL NAILS OF THE HANDS. Presented by DR. WISE.

Dr. B., a private patient, was first seen a week previously, with a ringworm infection of all the nails, the duration of which was six months. The question of the radical removal of all the nails had been considered, and the patient was presented to get an opinion as to the advisability of this or of some less radical method of treatment.

ONYCHOMYCOSIS. Presented by DR. HOWARD FOX.

A salesman, aged 29, had had the condition for eight years. He first came under observation five or six years previously, with every finger of the left hand affected. He had received five roentgen-ray treatments of one skin unit each. He had not had any treatment for five months, and three of the nails were apparently normal. On culture, *Monilia*, an organism of the yeast group, was found.

PEMPHIGUS FOLIACEUS. Presented by DR. MACKEE.

Mrs. N. C., a Jewish woman, aged 35, born in the United States, had an eruption which began behind the left ear and in a scar on the abdomen in the early part of 1926. Within a few months, the eruption had become generalized. When the patient was presented, there was a generalized eruption, particularly on the face, arms, abdomen, chest and legs. At a distance, the eruption appeared to be dermatitis exfoliativa. There was considerable erythema and what appeared to be dry exfoliation. On close examination, various sized bullae were noticed. These bullae were so flaccid and occurred so often under the scales that they were easily overlooked. There was a little exudation in places. It was possible to remove the apparently normal epidermis by slight friction. There were occasional tense bullae, but they never appeared on the unaltered skin. The patient was in excellent physical condition. An unusual feature was considerable itching. There were distinct remissions and exacerbations. With the exception of a tiny ulcer on the tip of the tongue, which had occurred several months previously, there had been no lesions in the mouth.

The patient did not tolerate arsenic well, even when combined with sodium thiosulphate. It always produced chills, fever and prostration. Every time arsenic was given, the eruption became worse. All sorts of treatment had been given. When presented, the patient's skin was in comparatively good condition. While many different forms of treatment had been given and while some of them, such as prolonged baths, ultraviolet radiation, oil baths and dry powder treatment, appeared to be of benefit, it was doubtful if any treatment had had any effect on the disease other than to make the patient a little more comfortable.

When presented, there were numerous new flaccid bullae which represented the beginning of an exacerbation.

The laboratory examinations revealed that the blood culture was negative. A chemical examination of the blood showed: sugar, 0.11 mg.; creatinine, 2.2

mg. per hundred cubic centimeters; urea nitrogen, 11.34 mg.; uric acid, 3.1 mg.; chlorides, 0.69 mg.; calcium, 3.3 mg. Urinalysis was negative. Results of examination of the stool and of the examination of the contents of the stomach were negative. The Wassermann reaction was negative. The blood count showed a slight secondary anemia.

In the general physical examination, the results of the basal metabolism test and roentgen-ray examination of the teeth, thorax, abdomen and pelvis were negative. The temperature was subnormal in the morning, and 99.5 or 100 F. at night by mouth. The pulse rate ranged between 80 and 100.

Dr. MacKee had decided to try blood transfusion.

DISCUSSION

Dr. Clark: I agree with the diagnosis. I do not see any lesions in the mouth. I would suggest the use of arsenic, which has always given the best results at the Skin and Cancer Hospital. At the suggestion of Dr. Throne and Dr. Myers, arsenic has been given by mouth and sodium thiosulphate by injections to eliminate the arsenic. This is a good way to eliminate arsenic when one desires to push the intake of arsenic.

Dr. Throne: Referring to Dr. Clark's statement about the administration of sodium thiosulphate in addition to arsenic: I know of a man with chronic pemphigus, who, with the addition of sodium thiosulphate, has taken from ten to sixteen Asiatic pills a day. This has kept him comfortable now for eighteen months. Some time ago I saw, in consultation, a woman with chronic recurrent pemphigus who had been given as many injections of iron cacodylate. The lesions would not heal, and the patient complained of intense pruritus. There was marked pigmentation around the lesions and the sites of the old lesions. The only treatment I could suggest was the administration of sodium thiosulphate to eliminate the arsenic, and to my surprise the pigmentation disappeared, the old lesions healed, and for some time the patient was free from symptoms of pemphigus.

Angioma Cavernosum Treated with Radium. Presented by Dr. MacKee.

A. N., a girl, aged 16 months, was normal at birth. A day or two after birth, a dime-sized strawberry mark was noticed on the chest. About five days later, a slightly elevated, red lesion about the size of a split pea appeared on the left eyelid. A few days later, a tiny lesion of similar character appeared on the left cheek. Then, suddenly, an enormous cavernous angioma developed over the entire face, neck and chest. The angioma was so extensive, so deep-seated and elevated, and involved so many structures, such as the mouth, nose, eyelids and ears, that it made a monstrosity of the infant. When first seen by the presenter, the infant was 4 months of age and badly emaciated. There was so much obstruction, because of pressure on the chest and throat, that breathing was difficult and rapid, and it was difficult for the child to take nourishment.

The patient had received radium treatment for about one year. Suberythema doses of beta rays had been used. No one area had received more than six or eight treatments, at intervals of six or eight weeks. The result was remarkable. The child was fat and in perfect health. Most of the angioma had disappeared. A number of areas required more treatment. There was considerable wrinkling of the skin in places, but this was probably chiefly, if not entirely, due to changes produced by the angioma.

Beta rays had been used because they sometimes produced the desired effect even in deep-seated angioma of the cavernous type, and when they did have the desired effect they were preferable to gamma rays because there was less danger of injuriously affecting the viscera, bones and other parts. Half strength, glazed, flat radium element applicators were employed. They were screened with 0.2 mm. of aluminum. They were held in contact with the skin for ten minutes as a rule. Some of the applications lasted for only five minutes. Photographs of the condition before treatment had been instituted were demonstrated.

Dr. MacKee stated that often it was impossible to obtain a result like this with beta radiation. In such instances it was necessary to use a filter of at least 1 mm. brass and 1 mm. aluminum.

DISCUSSION

Dr. Howard Fox: The result of the treatment is excellent, especially in view of the frightful appearance of the patient before treatment.

Dr. Whitehouse: It is a marvelous case. Most physicians would hesitate to treat the patient with radium.

Extensive Lupus Erythematosus Treated with Sodium Thiosulphate. Presented by Dr. Clark.

Mrs. C. A. S., a private patient, aged 47, born in the United States, first began to have lupus erythematosus of the face eight years before presentation. The patient claimed that it came on rather suddenly. These lesions spread and increased in number; new lesions appeared on the V-shaped area of the neck and on the forearms. When first seen, a year ago, almost the entire face of the patient was covered with a thick, infiltrated type of lupus erythematosus in large plaques, some scarred areas where other lesions apparently had healed, rather large plaques on the chest, and scattered plaques on the forearms.

During a period of eight years, the patient had been treated by most of the experienced dermatologists of the country. For a long time the lesions had been painted with iodine, and the patient was given quinine. Dr. Robert Abbe treated her with radium, with some apparent temporary success. Light treatments of all kinds, especially ultraviolet radiation, was used for long periods of time without apparent success. Lotio alba was applied for many months. Various strong ointments and even curetting had been resorted to, but the lesions persisted, and new lesions had gradually developed. Also, nonspecific protein injections were ineffectual.

The patient consulted Dr. Clark about a year before presentation, requesting applications of carbon dioxide snow; but as Dr. Schamberg had reported marked improvement in lupus erythematosus with his injections of gold protein, Dr. Clark secured some of this remedy from him. The patient began to show marked improvement after the second or third injection, but her constitutional reactions after the fourth or fifth treatment became so severe that Dr. Clark was afraid to continue the treatment, even after so remarkable an improvement.

Seven months before presentation, the patient began to receive gold thiosulphate in the form of a gold compound in doses of 50 mg., three times a week, and later, doses of 100 mg., twice a week. With a few interruptions she had continued these treatments for a period of five months, with a gradual but certain improvement of the lesions. When presented she had not had any treatment for a month, and her face was entirely clear, with a surprisingly

small amount of scarring, except for the original scars which were present when she first presented herself and some redness of the tip of the nose with rather pronounced dilated blood vessels. The lesions on the neck and on the forearms had cleared without scarring.

The patient was presented because the condition was one of the most pronounced and extensive cases of infiltrated lupus erythematosus ever observed by the presenter, and in his opinion could not have been cleared up with any form of treatment other than the gold thiosulphate, which has proved to be almost a specific in the treatment of lupus erythematosus.

DISCUSSION

DR. WISE: I have had uniformly good results with gold and sodium thiosulphate and with a gold compound in the few patients I have treated. One of the patients had an extensive dermatitis, but the condition cleared up within a week after the injection of six doses of gold and sodium thiosulphate.

DR. THRONE: I observed a series of twenty-five patients who were treated with injections of a gold compound in connection with gold and sodium chloride by mouth. Five of them had reactions varying from pruritus up to a widespread erythema. Three of them showed focal infection. There were six other patients who showed focal infection and who were removed before treatment was instituted. None of these showed any reaction. The reactions from the gold seemed to run parallel with the reactions from the arsphenamine described by Stokes. It has seemed best in our clinic to have the patients examined for local infection before instituting the radical treatment.

DR. WAY: Hoffmann has had excellent results with the use of gold preparations in cases of lupus erythematosus. The results were much better when the patients were treated at the same time with carbon arc radiation. He also found that the patients responded better to mild doses of the medicine when given in low dilution and increased gradually. He had had some cases in which fever developed when he increased the doses too rapidly.

DR. THRONE: All the patients I spoke of as having focal infection had severe dental infection.

DR. BECHET: There are three distinct types of reaction: (1) constitutional (malaise, chill, fever, prostration), (2) a generalized dermatitis of an erythematovesicular type and (3) a local reaction in the lesions themselves, characterized by an increase in the inflammation with sensations of burning and itching. I have observed local reactions frequently, and the more active this reaction the more rapid is the improvement. I have seen only one case in about forty in which there was eczematoid dermatitis without constitutional reactions.

DR. MACKEE: I have seen several instances of systemic reactions to gold in patients in whom it was impossible to detect a focus of infection. I have had many excellent results with the gold treatment in severe cases of lupus erythematosus. Recently I saw a severe focal reaction after a few injections. At first there were only two or three small, rather inconspicuous patches of discoid lupus erythematosus. After two or three injections, the eruptions changed to the disseminate type, and spread all over the face, neck, forearms and hands. The man was given sodium thiosulphate, and is now again receiving the gold treatment but with extreme caution. The condition is beginning to improve. Not infrequently a patient who at first does not tolerate gold very well, will later stand large doses.

Any kind of radiation must be used cautiously in lupus erythematosus. Goeckermann, Wise and I have reported examples of the wide spreading of lupus following erythema doses of ultraviolet radiation applied, not to the lesions, but to the glandbearing regions. In one instance, lupus erythematosus appeared on the chest and back where a suberythema dose of roentgen rays had been applied.

DR. BECHET: I wish to corroborate what Dr. MacKee said about the ultraviolet rays. In an old chronic discoid type of lupus erythematosus, the water-cooled quartz lamp with compression is a good destructive agent, but distance radiation with the air-cooled lamp is occasionally followed by an extension of the disease. This was well illustrated in a case reported by me in which the use of an air-cooled lamp on a discoid lupus erythematosus was followed by rapid extension of the disease in the areas of actinic erythema, resulting from the single treatment. I should, therefore, advise considerable caution in its use.

LUPUS ERYTHEMATOSUS TREATED WITH GOLD AND SODIUM THIOSULPHATE. Presented by DR. CLARK.

Miss C. C., aged 40, born in Ireland, a housewife, when first seen, two years previously, had a typical discoid lupus erythematosus of the infiltrated, thick variety, involving the tip of the nose, the nasal septum and the upper lip below the nares, extending down on to the vermilion border of the lip.

The patient had received many kinds of treatment without improvement, and during the first year or so she had all her infected teeth removed and her intestinal tract put in good condition by diet and medication, with negative results.

Five months before presentation, gold thiosulphate treatment was started in the form of a gold compound, beginning with doses of 50 mg., three times a week, and gradually increasing to doses of 100 mg., twice a week. There had been a marked improvement of the lesions of the face. In the meantime several flat, macular lesions that were faintly red appeared on the lower part of both thighs. Two weeks later, these lesions became somewhat infiltrated, and a scale developed. The administration of gold thiosulphate was stopped and sodium thiosulphate injections were instituted in the belief that this might be an eruption from the gold thiosulphate, but the lesions persisted and became covered with a distinct black scale, on the under surfaces of which were little horny plugs.

Treatment with gold preparations both by mouth and by injection, had failed to influence the lesions on the legs.

The case was presented to show a practical cure of the lupus erythematosus of the face, and for diagnosis of the lesions on the legs.

DISCUSSION

DR. MACKEE: I think that the condition is lupus erythematosus.

DR. THRONE: I have seen one case like this. The primary lesion was on the nose and adjacent parts of the cheeks. Later, other lesions developed on the chest during treatment.

DR. ELLER: I also agree with the diagnosis of lupus erythematosus for the new lesions on the thighs. This case is interesting because of the appearance of these lesions during the time of treatment of the lupus lesions on the face with injections of gold.

TUBERCULOSIS VERRUCOSA CUTIS. (TWO PATIENTS.) Presented by DR. WILLIAMS.

CASE 1.—Miss H. S., aged 23, a stenographer, about a year prior to presentation had an operation on the dorsal surface of the right hand for removal of a tumor attached to the tendon. On examination, this proved to be a tuberculoma. The eruption for which the patient was presented began as a callous spot on the left index finger five or six months previously. This was treated by the patient with repeated doses of iodine. Other similar small callous spots appeared and were treated in the same way. They became infected, discharging small amounts of pus. Later, painless, fluctuating swellings appeared on the dorsal surface of the second phalanx of the left index finger. The inflammation subsided under treatment with wet dressings of boric acid, revealing three small spots hardly distinguishable from calosities and one larger spot in which there was a distinct loss of tissue, and also some apparent hypertrophy of the papillae. The patient was being treated with ultraviolet light.

CASE 2.—R. F., a girl, aged 8, born in the United States had a condition which began in August, 1926, as a callous spot. There were now two callous-like lesions with slight necrosis in the center on the palmar surface of the first phalanx of the left index finger. Hypertrophy of papillae was not evident.

DISCUSSION

DR. HIGHMAN: I think that the woman has tuberculosis verrucosa cutis; the little girl has a wart. In both cases a microscopic examination should be made.

DR. WISE: I agree with Dr. Highman that the older patient has tuberculosis verrucosa cutis, and that the lesion of the little girl is more like an ordinary wart.

DR. MACKEE: I hesitate to question the diagnosis without giving a constructive suggestion. In the case of the woman, the eruption does not appear to be sufficiently thick, deep and verrucous for warty tuberculosis. It is probably some form of tuberculosis cutis. I should think it would be necessary to exclude verruca vulgaris in the case of the little girl.

DR. LANE: I think that the older patient had tuberculosis verrucosa cutis. I should hesitate to do a biopsy on that finger just now. There is active tuberculosis in the tendon sheaths.

DR. WHITEHOUSE: I consider that Dr. Williams' remarks in regard to the tuberculous history in the older patient would make the diagnosis of tuberculosis of the skin probable. In regard to the child, I agree that the condition is probably an inflamed verruca vulgaris.

DR. WILLIAMS: The older woman has a tuberculous inflammation of the tendon as proved by pathologic examination, and I believe the keratotic growths are produced by the same organism. An inflammation produced by the ordinary pus organisms should be more acute and painful. The only treatment has been sulphonated bitumen, N. F., boric acid and ultraviolet light. Although the lesions look like warts at the start, as they develop suppuration and loss of tissue occur.

The lesions on the child are perhaps less suggestive of tuberculosis than those on the woman, but they are distinctly inflammatory.

I would not care to risk a biopsy on the young woman. I shall endeavor to secure a biopsy on the child and report later. Of course, neither case is typical of tuberculosis verrucosa cutis; had the cases been typical, I would not have presented the patients.

ONYCHOMYCOSIS SUCCESSFULLY TREATED BY ROENTGEN RAY. Presented by DR. HOWARD FOX.

I. J., aged 31, a traveling salesman, had suffered from the eruption for eight years. Previous to roentgen-ray treatment all the nails of the left hand had been affected; the nails of the right hand and of the toes were normal. The patient had previously been treated for a year by scraping followed by vigorous inunctions with salicylic acid, without the slightest result. The entire nail of each finger was lusterless, discolored and rough from erosion of the outer table. There was no evidence of paronychia. Microscopic examination showed the presence of fungus, and cultures on two occasions showed a yeastlike organism, probably *Monilia*.

On March 22, 1926, roentgen-ray treatment was begun, consisting of unfiltered irradiation given on three occasions at monthly intervals to each nail, and on two subsequent occasions at intervals of two months; the dosage was one skin unit applied to the nail, the surrounding skin being closely screened. Ten months after the first treatment, three of the nails were entirely well.

DISCUSSION

DR. RULISON: The result is excellent. I am rather inclined to think there will be a recurrence if roentgen-ray treatment is depended on. The treatment that I have been using for these finger-nails, and which seems to be the most successful, is a wet dressing of strong mercuric chloride for several hours a day, the length of treatment being gradually increased as the skin will stand it. That treatment continued for two or three months seems to clear up most cases, and, as far as I know, permanently.

DR. WILLIAMS: The finding of an organism of the yeast group in the nails is exceedingly interesting. Cases like this usually were thought to belong in the group of trichophytoses, but there is no reason why there should not be a yeast organism. I had a similar case of onychomycosis of the fingers and toes, and also infection intertrigo of the fingers. Scrapings showed a yeastlike organism. This was confirmed by culture. It happened that this patient was sent to me by a patient with onychomycosis whom I had previously treated with the roentgen ray and light; as I have not seen her for some time and as she referred this woman to me, I presume she was cured. I have had no success with antiseptics, but I have had some good results with the roentgen rays and ultraviolet light.

DR. WISE: The case brings to mind a suggestion that infections of the nails due to yeast organism respond better to the roentgen ray than do ringworm infections.

DR. MACKEE: I have had three cases of onychomycosis in which *Monilia* was demonstrated in cultures. In answer to Dr. Lane's question, it has been demonstrated in the laboratory that roentgen rays and gamma rays of radium do not have any direct effect on the fungi. Whatever effect is obtained is probably the result of a changed environment. This type of irradiation appears to be able to make the environment less favorable to the growth of organisms. The beta rays of radium are able to kill bacteria and fungi, but, according to my recollection, the dose required is larger than that used on human beings.

DR. HOWARD FOX: Enough patients with onychomycosis have been successfully treated by the roentgen ray to prove that the results are not merely a coincidence. It is true that the treatment is often unsuccessful. How the rays act is not known; it is certain that they are not bactericidal or fungicidal.

Possibly, as Sabouraud suggests, they cause some change in the keratin which makes it an unsuitable soil for the fungus. I was interested to hear the suggestion that diseases of the nail due to yeast are perhaps more amenable to treatment than infection due to mold. The cultures in my case showed yeast-like organisms on two occasions.

DERMATOLYSIS (MOLLUSCUM TYPE). Presented by DR. MACKEE.

Mr. H. K., aged 20, unmarried, had an eruption of unknown duration. He thought it had always been present. The condition was limited to the lower dorsal and lumbar regions. The area consisted of an irregular band about 5 cm. wide and about 20 cm. long. The skin in this area was rather spongy and slightly elevated; the line markings were accentuated, and the follicles were exceedingly patulous. This gave the skin a coarse appearance. The color was a little darker than the patient's normal skin. On palpation, the skin felt soft and spongy. There were not any subjective symptoms.

Attention was called to the fact that this was an exact duplicate of the case published by Wise in 1914. Dr. MacKee also presented photographs of several cases of this type of dermatolysis with lesions about the size of a quarter scattered over the upper part of the back. The pictures were taken of several different patients, two of whom also had syphilis. The presenter called attention to the fact that with the exception of the location and the size of the lesions, the cases were exactly the same. Microscopic examinations made on some of the cases showed myxomatous tissue.

BROMODERMA TREATED WITH SODIUM CHLORIDE. Presented by DR. MACKEE.

Mr. P. R., aged 29, unmarried, had been taking bromides for over a year. Shortly after beginning to take the medicine, a lesion appeared on the outer aspect of the right leg, just above the ankle. This lesion spread until it acquired the size of a man's palm. It was dull red, elevated about 1 cm., and was verrucous. Bromides had been administered for epilepsy and other nervous disorders. The drug was discontinued and phenobarbital was substituted. The lesion disappeared. Later it became necessary again to administer bromide. The lesion promptly recurred. It had been impossible to dispense with bromide. The patient took from 60 to 120 grains (3.91 to 7.8 Gm.) of sodium bromide daily. The lesion, however, had almost disappeared, even while the patient was taking his daily doses of bromide, as a result of the administration of liberal doses of sodium chloride by mouth. The patient used considerable salt in his food, and in addition he took one teaspoonful of table salt in a little water daily. When presented, there was a slightly verrucous skin over an area the size of a silver dollar, with no elevation above the surface.

DISCUSSION

DR. WISE: Phenobarbital is often used as a substitute for bromides in cases in which bromoderma becomes an obstinate lesion.

DR. WHITEHOUSE: I would like to testify as to the control of epilepsy by phenobarbital. I had a patient who had attacks every day, and phenobarbital helped him so that he did not have them for several months.

DR. BECHET: The fact that the administration of salt by mouth might arrest or even ameliorate an existing bromoderma seems demonstrated in Dr. MacKee's case. I am glad of the opportunity of seeing the patient, as there have been some references to the possible good effect of salt by mouth in bromoderma. Recently I had under observation a girl who had taken a gastric tonic con-

taining bromide; within a month she developed a severe bromide acne. A teaspoonful of salt, morning and night, and a plentiful supply of salt in her food had a miraculous effect on the bromoderma, the lesions disappearing within a few weeks. Of course, the results following the oral administration of salt cannot compare with the results when intravenous injections are given; in my hands the latter fully corroborate the results obtained and reported by Wile.

DR. THRONE: Bromoderma sometimes occurs in infants who could not be given intravenous injections. The ingestion of salt might be helpful.

DR. HOWARD FOX: I am glad this matter has been brought to the attention of the Society. In cases in which it is apparently necessary to administer bromides over a long period, it is more convenient for the patient to take salt by mouth than to have intravenous injections of salt solution, as recommended by Wile in the treatment of bromoderma.

LEPROSY. Presented by DR. WISE.

N. K., a man, aged 42, a butcher, was presented before this Society about four years previously. Dr. Lane confirmed the diagnosis by biopsy five years previously, and under treatment with injections of ethyl ester of chaulmoogra oil the eruption cleared up, but in September, 1926, it recurred. When presented the second time there were lesions on the chest, thighs, face, arms and shoulders. The lesions were macular, violaceous and, for the most part, circinate and discoid. On the face they were of a brownish-yellow hue, while on the trunk the color varied from faint yellow to dark purple tints. Subjective symptoms were absent. These lesions made their appearance while the patient was under active treatment.

NODULAR LEPROSY. Presented by DR. HOWARD FOX.

V. J. T., aged 37, born in the Philippine Islands, a cook, came to the United States thirteen years previously. With the exception of a single short trip to the West Indies, he had lived continuously in or about New York. Without any premonitory symptoms the eruption suddenly appeared about six months before presentation as reddish-brown, discrete, nodular masses over the chin, cheeks, parotid region, left ear and right instep. Lepra bacilli were not present in nasal smears on two occasions, but were found in excised tissue. He was being treated with chaulmoogra oil and carbon dioxide snow.

GRANULOMA OF UNKNOWN TYPE. RESULT OF ROENTGEN-RAY TREATMENT. Presented by DR. WISE.

H. W. F., aged 32, was previously presented on several occasions (Manhattan Dermatological Society, January, 1926, ARCH. DERMAT. & SYPH. **13**:844 [June] 1926; also Atlantic City Conference, Dec. 16, 1925, ARCH. DERMAT. & SYPH. **13**:603 [May] 1926). Roentgenotherapy was administered by means of filtered massive dosage at intervals of from six to eight weeks, resulting in striking improvement of the hypertrophic growths in the groins and inguinal regions. These had become absorbed to at least 75 per cent of their former size; the areas had flattened and become smooth and free from vegetations.

ANGIOLUPOID OF BLOCH AND PAUTRIER? Presented by DR. THRONE.

Mrs. A. R., aged 34, Jewish, had had a condition of nine months' duration. The patient showed an oval lesion on the right side of the nose, which extended

obliquely downward and outward onto the cheek. The dimensions of the lesion were 13 mm. by 5 mm.; its elevation was about 2 mm. The color was brownish red. On diascopic pressure, a brownish-yellow tint was left. This color was uniform. The consistency was soft. The epidermis was apparently thinned. There had never been any scaling.

The patient was given many injections of a gold preparation, without benefit. She was being treated with roentgen rays. There had been some lessening of the infiltration since this treatment was instituted. A biopsy was not allowed.

NEVUS VERRUCOSUS. Presented by DR. BECHET.

A. V., a girl, aged 11, since birth had had an extensive hyperkeratotic, papillomatous and pigmented nevus, covering practically the entire left side of her body from the neck down to the calf.

NEVUS VERRUCOSUS. Presented by DR. BECHET.

D. L., a boy, aged 12, presented large bilateral, pigmented, papillary hyperkeratotic bands, arranged geometrically over the entire body. The lesions had been present since birth.

DEFORMING SCARS FOLLOWING UNSUCCESSFUL PLASTIC OPERATION. Presented by DR. HOWARD FOX.

Miss M. C., aged 36, a nurse, first applied for treatment of an epithelioma of the nose seven years prior to presentation. She had previously received treatment for this tumor, but it had recurred. A second recurrence took place two years later; she was again treated, and had been free from the disease since then. A perforation of the nose near the tip, leading into the nasal cavity remained. An effort to insert an ivory transplant by surgical procedures failed. Later another surgeon attempted to relieve the deformity by flap operation, making one attempt to obtain a flap from the forehead and another to obtain a flap from the side of the neck. Both attempts were unsuccessful, and were followed by extensive deforming scars, one on the left side of the neck and chest being 10 inches (25.4 cm.) in length and as disfiguring in places as scars of a former scleroderma. The patient apparently had never suffered from syphilis.

DISCUSSION

DR. CLARK: This should not be accepted as a usual result of plastic surgery. There have been remarkable results, particularly in some of the operations performed by Dr. Semken and Dr. H. H. M. Lyle in cases of saddle back deformity. This is just a single bad result, and it does not mean that treatment by proper plastic surgery should not be attempted in these cases. I think Dr. Throne will bear me out in that.

DR. FOX: I presented the patient merely to show severe scarring resulting from operation to relieve a comparatively slight deformity. I did not mean to disparage the results of cosmetic surgery in general.

HAT BAND DERMATITIS. Presented by DR. BECHET.

P. R., a man, aged 50, from the service of Dr. Whitehouse, said that the eruption had been present for twelve months. He was almost bald, and on the bald spot over the upper occipital region and on the forehead was an erythematous patch, dry but not scaly, arranged in a bandlike formation. On

putting on his hat, the eruption was exactly covered by the hat-band, both in front and behind. It did not occur on the parts covered with hair, nor on any other part of the body. He said that in summer, when he wore his hat less, the bandlike patch of eruption markedly improved, but that it increased again with more frequent use of the hat.

MYCOSIS FUNGOIDES AND MIKULICZ'S DISEASE. Presented by DR. THRONE.

M. B., a man, aged 49, from the skin and cancer hospital, born in the United States, began to have pain and swelling of both parotids about two months previously. The swelling gradually became more marked. Later rounded red patches developed on the chest, back and limbs. These patches had never itched. His general health had always been good, except for indigestion and stomach trouble. The patient claimed that all his disorders began when he was being treated for his stomach condition. When presented, the parotid glands were very large and hard. There were dull red, slightly infiltrated plaques scattered over the body, varying in size from that of a quarter to that of a palm. The cervical, axillary and inguinal nodes were slightly enlarged. The spleen was not palpable. There had not been any pruritus. Some of the patches showed acuminate, follicular miliary lichenoid lesions. The biopsy report is not yet ready. The blood count revealed: red cells, 4,910,000; white cells, 11,000; polymorphonuclears, 89 per cent; lymphocytes, 20 per cent; mononuclears, 1 per cent; transitionals, 1 per cent; eosinophils, 1 per cent, and basophils, 1 per cent.

LICHEN PLANUS. Presented by DR. MALONEY.

A. D., a man, aged 63, a mulatto whose father was white and whose mother was colored, was born in Florida, and was a street cleaner. He complained of severe itching which had endured for four months. Examination showed numerous discrete lesions varying in size from that of a match-head to that of a dime, situated mostly on the buttocks and lower extremities, with a few lesions on the forearms. The primary lesion appeared as a small red prurigo inous papule which increased in size, the center becoming bluish black and depressed, and apparently showing atrophic and sclerotic changes. The larger lesions and patches were surrounded by an areola and had a ringed appearance. There were no lesions of the mucous membrane. The blood Wassermann reaction was negative. Albumin or sugar was not found in the urine. The blood count showed leukocytes, 8,600; large lymphocytes, 12 per cent; small lymphocytes, 15 per cent. A microscopic examination of a section taken from one of the lesions showed the picture of lichen planus.

JOHN E. LANE, M.D., Secretary

Book Review

EMINENT CHEMISTS OF OUR TIME. By BENJAMIN HARROW. Second Edition. Pp. 470. New York: D. Van Nostrand Company, 1927.

This book contains a series of brief biographies of the most eminent chemists of the present time, men who have taken an outstanding part in the phenomenal development of chemistry since Perkin produced the first aniline dye in 1856. There are sketches of Perkin, Mendeleeff, Ramsay, Richards, van't Hoff, Arrhenius, Moissan, Madame Curie, Victor Meyere, Remsen and Emil Fischer. In the second part of the work, which is an addition to the second edition, there is a brief analysis and bibliography of each one's work. Harrow has not lost the spirit of youth, and he writes of these masters in his field with enthusiastic appreciation and vivacity. There is nothing more stimulating to a man than the biographies of the prominent workers in his field. This book can be recommended to any one who wants to whet his appetite for scientific research. The sketches are vivid; most of the space is given to achievements, but they contain enough of the personal touch to give them satisfactory human interest and to make one realize that one is dealing with men and not with imaginary demigods. There is little variation in the quality of the sketches; they are all good; so good in fact that the reviewer, since reading the book, has run across an address in a scientific journal on the life of one of the subjects of this work, much of which has been lifted bodily from Harrow's sketch without giving credit to this author. The work can be recommended to any one who has any scientific interest, whether he is a chemist or not. The careers of these men are inspiring examples of the careers of great scientific investigators in general.

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Index to Current Literature

DERMATOLOGY

Acanthosis Nigricans. M. Takuwa, J. Kyoto M. Soc. **1**:34 (July) 1927.
Active Immunization of a Community to Scarlet Fever and Diphtheria. B. de Rudder, München med. Wchnschr. **74**:1357 (Aug. 12) 1927.
Agglutinins in Human Serums for Scarlet Fever Streptococci. L. Baumgartner, Am. J. Pub. Health **17**:814 (Aug.) 1927.

Bacteriologic Studies of Impetigo: I. Impetigo Vulgaris. M. Aochi, Acta derm. **9**:539 (June) 1927.

Capacity of Dermatology. W. A. Pusey, New York State J. Med. **27**:828 (Aug. 1) 1927.
Case of Chronic Dermatomyositis. M. J. Carney, Canad. M. A. J. **17**:911 (Aug.) 1927.
Case of Pellagra. E. B. White, J. Ment. Sc. **73**:430 (July) 1927.
Case of Pellagra with Recurring Attacks. W. J. T. Kimber, J. Ment. Sc. **73**:433 (July) 1927.
Case of Pityriasis Rubra with Unusual Course. W. Pospeloff and D. Krupnikoff, Acta derm.-venereol. **8**:59 (June) 1927.
Chickenpox Orchitis. J. Sabrazès, Bull. Acad. de méd. **98**:122 (July 26) 1927.
Chronic Purpura with Thrombopenia. H. H. Lautier et al., Rev. franç. de pédiat. **3**:341 (July) 1927.
Coccidioidal Granuloma, I. D. Reisman and F. E. Ahlfeldt, Am. J. M. Sc. **174**:151 (Aug.) 1927.
Connection Between Vulvitis Aphthosa and Erythema Nodosum. E. Schnabl, Dermat. Wchnschr. **85**:1281 (Sept. 10) 1927.
Control of German Measles and Chickenpox. C. W. Gerber, Southwestern Med. **11**:400 (Sept.) 1927.
Cytology of Molluscum Contagiosum. E. W. Goodpasture and H. King, Am. J. Path. **3**:385 (July) 1927.

Dermo-Elastometry. F. Schmidt-Labaume, Dermat. Wchnschr. **85**:1217 (Sept. 3) 1927.
Diagnosis of Skin Tumors. E. Balbi, Arch. di biol. **4**:41 (May-June) 1927.
Diathermy Treatment of Roentgen Ray Ulcerations. H. Bordier, Acta radiol. **8**:189 (July 30) 1927.
Dick Test in Relation to Presence of Streptococcus Hemolyticus in Throats of Individuals not Suffering from Scarlet Fever. J. Smith, Arch. Dis. Child. **2**:166 (June) 1927.
Diphtheria and Scarlet Fever Immunization. J. P. Kinloch, J. Smith and J. S. Taylor, J. Hyg. **26**:327 (Aug.) 1927.
Dystrophy of Skin and Muscles of Nervous Origin. T. B. Wernøe, Ugesk. f. Laeger. **89**:709 (Aug. 11) 1927.

Effect of Circulation on Electrical Resistance of Skin and Man. H. M. Wells, Quart J. Exper. Physiol. **18**:33 (July 13) 1927.
Emotional Blushing. P. Hartenberg, Presse méd. **35**:1003 (Aug. 13) 1927.
Epidemic of Roseola Infantum. H. B. Cushing, Canad. M. A. J. **17**:905 (Aug.) 1927.
Epidemic Pemphigus of Infants. L. Garot, Rev. franç. de pédiat. **3**:353 (July) 1927.
Epidermophytosis. H. Odland, Northwest Med. **26**:387 (Aug.) 1927.
Erythema Action of Various Qualities of Roentgen Rays. A. Determann et al., Strahlentherapie **26**:472, 1927.
Erythema Nodosum. R. Massini, Schweiz. med. Wchnschr. **57**:708 (July 23) 1927.
Erythema Nodosum and Pulmonary Tuberculosis. C. Hambro, Acta tuberc. Scandinav. **3**:1, 1927.
Erythema Simulating Scarlatina. T. F. Anderson, Kenya & E. African M. J. **4**:113 (July) 1927.

Etiology of Scarlet Fever. A. Smirnowa-Zamkowa, Virchows Arch. f. path. Anat. **265**:1 (July 6) 1927.
Experimental Malignant Ulcers in Skin. M. T. Burrows, Arch. Path. **4**:380 (Sept.) 1927.

Fungus Disease of Skin. J. R. Allison, South. M. J. **20**:604 (Aug.) 1927.

Gas Gangrene as Sequela of Erysipelas. K. Marcuse, Zentralbl. f. Chir. **54**:1867 (July 23) 1927.
Gland Puncture Findings in Leprosy. J. M. Henderson, Indian M. Gaz. **62**:438 (Aug.) 1927.

Hemolytic Streptococcus Carriers: Their Relation to the Spread of Scarlet Fever. Mary B. Kirkbride and Mary W. Wheeler, J. A. M. A. **89**:1394 (Oct. 22) 1927.
Hemophilia with Purpura. W. T. Fotheringham and B. Palamedi, Semana méd. **34**:157 (July 21) 1927.
Henoch's Purpura: Case. R. W. Bradshaw, Ohio State M. J. **23**:754 (Sept.) 1927.
Herpes Simplex and Zoster. H. Freund and B. Heymann, Ztschr. f. Hyg. u. Infektionskrankh. **107**:592 (July 25) 1927.
Herpes Zoster with Paralysis of Abdominal Muscles and Postherpetic Neuralgia. M. G. Masten, Arch. Neurol. & Psychiat. **18**:437 (Sept.) 1927.

Increased Penetrability of Skin for Short Light Waves and Ultraviolet Rays. W. E. Pauli, Strahlentherapie **26**:577, 1927.
Infectious Exanthem of Undetermined Nature. Bezançon et al., Bull. Acad. de méd. **98**:59 (July 19) 1927.
Islands of Squamous Epithelium in Cervical Mucous Membrane. H. Hinselmann, Monatschr. f. Geburtsh. u. Gynäk. **76**:442 (July) 1927.

Kala-Azar: Demonstration of Leishmania Donovani in the Skin and Subcutaneous Tissue of Patients: Possible Relation to the Transmission of the Disease. J. R. Cash and C. H. Hu, J. A. M. A. **89**:1576 (Nov. 5) 1927.
Kala-Azar in Simla Hills. P. Savage, Indian Med. Gaz. **62**:382 (July) 1927.

Lactic Acid Formation in Scarlet Fever Streptococci and Ordinary Pus Streptococci. A. Kappus, Ztschr. f. Hyg. u. Infectionskrankh. **107**:697 (July 25) 1927.
Leprosy on Bengal-Bihar Border. E. Muir, Indian M. Gaz. **62**:440 (Aug.) 1927.
Leprosy Survey at Manbazar Thana, District Manbhum, Bihar and Orissa. I. Santra, Indian M. Gaz. **62**:442 (Aug.) 1927.
Leukoderma and Melanoderma in Skin Diseases. J. Almkvist, Acta derm. Venereol. **8**:91 (June) 1927.
Local Amyloidosis of Skin. C. Gutmann, Dermat. Wchnschr. **85**:1223 (Sept. 3) 1927.
Local Amyloidosis of Skin. C. Gutmann, Dermat. Wchnschr. **85**:1283 (Sept. 10) 1927.
Long Standing Granuloma Inguinale. F. W. Rice, Am. J. Obst. & Gynec. **14**:249 (Aug.) 1927.
Lupus Vulgaris in Arizona. N. D. Brayton, Southwestern Med. **11**:357 (Aug.) 1927.

Madura Foot in Georgia Negro. C. C. Harrold, South. M. J. **20**:654 (Aug.) 1927.
Malignant Degeneration of Calcified Epithelioma of Skin. N. Gromiko, Virchows Arch. f. path. Anat. **265**:103 (July 6) 1927.
Management of Carbuncles. A. D. Wilmoth, Kentucky M. J. **25**:414 (Aug.) 1927.
Management of Measles in Hospital and Home. H. J. Spencer, J. A. M. A. **89**:1662 (Nov. 12) 1927.
Measles: Prophylactic Use of Convalescent Whole Blood. G. S. Buttorff, Kentucky M. J. **25**:406 (Aug.) 1927.
Measles Prophylaxis by Use of Parent's Blood Serum. L. Bivings, South. M. J. **20**:735 (Sept.) 1927.
Measles Simulating Scarlet Fever. J. Wilson, Brit. M. J. **2**:217 (Aug. 6) 1927.

Mechanism by Which Electrical Resistance of Skin is Altered. H. B. Densham and H. M. Wells, Quart. J. Exper. Physiol. **18**:175 (July 13) 1927.
Mental Aspect of Leprosy. L. L. Cazenavette, J. A. M. A. **89**:1496 (Oct. 29) 1927.
Modified Measles: The Use of Convalescent Blood from a "Family Donor." R. P. Forbes and B. Green, J. A. M. A. **89**:1601 (Nov. 5) 1927.

Naevus Pigmentosus of Scalp in Form of Cutis Capitis Gyrata. P. Gohrbandt, Arch. f. klin. Chir. **146**:644 (June 18) 1927.
Nature of Toxic Moiety of Streptococcous Scarlatinae. C. W. Duval and R. J. Hibbard, J. Exper. Med. **46**:379 (Aug. 1) 1927.
New Concentrated Scarlet Fever Serum. K. Ochsenius, München. med. Wchnschr **74**:1359 (Aug. 12) 1927.

Origin of Congenital Skin Defects. K. Lundwall, Arch. f. Gynäk. **130**:487 (July 28) 1927.
Osteosclerotic Anemia Secondary to Epidermoid Carcinoma. G. Y. Rusk and W. L. Miles, Am. J. Path. **3**:289 (July) 1927.

Patchy Pigmentation of Skin After Application of Heat. A. Buschke and M. Michael, Deutsche med. Wchnschr. **53**:1343 (Aug. 5) 1927.
Pathogenesis and Pathologic Physiology of Herpes Zoster. G. Marinesco and S. Draganesco, Presse méd. **35**:1025 (Aug. 20) 1927.
Pellagra. E. L. Bishop, J. Tennessee M. A. **20**:131 (Aug.) 1927.
Pemphigus in Thirteen Year Old Child. E. A. Morgan, Canad. M. A. J. **17**:1040 (Sept.) 1927.
Pemphigus Vegetans. A. E. Ruete, Deutsche med. Wchnschr. **53**:1301 (July 29) 1927.
Phagocytosis of Epithelium. S. Ishikawa, Acta derm. **9**:550 (June) 1927.
Pigeon Hole Dermatology. W. J. Highman, New York State J. Med. **27**:907 (Aug. 15) 1927.
Poikilodermia Atrophicans Vascularis. H. Geisler, Dermat. Wchnschr. **85**:1133 (Aug. 13) 1927.
Poisoning from Shoe Dye. L. Shinabery, Indiana State Med. Assoc. J. **20**:296 (Aug.) 1927.
Pollen Antigens, as Determined by Skin Reaction. R. F. E. Stier and G. L. Hollister, J. Lab. & Clin. Med. **12**:1139 (Sept.) 1927.
Protein Therapy in Skin Diseases. O. Michaëlis, Acta derm.-venereol. **8**:72 (June) 1927.
Psoriasis Treated with Injections of Thymus Solution. F. F. Ward, M. J. & Record **126**:216 (Aug. 17) 1927.

Relation of Thiersch Skin Transplant to Blood Groups. K. Matsuda, Arch. f. Japan. Chir. **4**:1 (July 20) 1927.
Relationship Between Scarlet Fever and Chronic Nephritis. D. G. Campbell, Canad. M. A. J. **17**:779 (July) 1927.
Results of Operations on Sympathetic in Scleroderma and in Dermatitis. R. Leriche and R. Fontaine, Rev. de chir. **65**:285, 1927.

Sarcomatosis Cutis and Mycosis Fungoides by Roentgen Rays. A. Whitfield, Lancet **2**:328 (Aug. 13) 1927.
Scabies. R. Blosser, Rhode Island M. J. **10**:134 (Sept.) 1927.
Scarlatinal Rheumatism Developing with Simple Endocarditis. E. Boltanski, Paris méd. **64**:83 (July 23) 1927.
Scarlet Fever in First Year of Life. F. von Bormann, Klin. Wchnschr. **6**:1615 (Aug. 20) 1927.
Scarlet Fever Protective Serum. F. von Bormann, Deutsche med. Wchnschr. **53**:1342 (Aug. 5) 1927.
Serum Test for Kala-Azar. R. N. Chopra, J. C. Gupta and N. K. Basu, Indian M. Gaz. **62**:434 (Aug.) 1927.
Skin Reactions. A. Bachem, Radiology **9**:241 (Sept.) 1927.
Sodium Hyposulphite Administered Intravenously in "Sanocrysin" Dermatitis. O. Jersild, Acta tuberc. Scandinav. **2**:361, 1927.
Specific Treatment of Scarlet Fever. H. H. Lautier and P. Dreyfus, Rev. franç. de pédiat. **3**:365 (July) 1927.
Streptococci in Throats of Scarlet Fever Convalescents. M. I. Kanevskaja, Mikrobiol. J. **5**:49, 1927.

Subacute Bacterial Endocarditis with Vesiculobullous Skin Lesions. D. Davis and D. Ayman, Am. Heart J. **2**:671 (Aug.) 1927.

Systemic Treatment of Skin Diseases. E. W. Abramowitz, New York State J Med. **27**:877 (Aug. 15) 1927.

Temperature of Skin. A. Seligman, Médecine **8**:752 (July) 1927.

Test for Kala-Azar. L. E. Napier, Indian M. Gaz. **62**:362 (July) 1927.

Transmission of Leishmania Tropica from Artificially Infected Sandflies to Man. S. Adler and O. Theodor, Ann. Trop. Med. **21**:89 (July 22) 1927.

Treatment of Asthmatic Patients Affected by Protein of Epidermal Group. Z. W. Stewart, J. Lab. & Clin. Med. **12**:1179 (Sept.) 1927.

Treatment of Eczema of Nipple and of Anogenital Region. A. Brandweiner, Wien. klin. Wchnschr. **40**:1035 (Aug. 11) 1927.

Treatment of Furuncles of Upper Lip. J. Sénèque, Presse méd. **35**:925 (July 23) 1927.

Treatment of Furunculosis by Diathermy. J. Hunter, J. Laryn. & Otol. **42**:524 (Aug.) 1927.

Treatment of Herpes by Intravenous Injections of "Sulphur" Water. R. Bénard and E. Joltrain, Bull. et mém. Soc. méd. d. hôp. de Paris **43**:1013 (July 7) 1927.

Treatment of Lupus Erythematosus by Krysolgan. H. C. G. Semon, Brit. M. J. **2**:258 (Aug. 13) 1927.

Treatment of Ringworm of Scalp by Thallium Depilation. G. B. Dowling, Brit. M. J. **2**:261 (Aug. 13) 1927.

Treatment of Superficial Burns. I. S. Ravdin, Atlantic M. J. **30**:679 (Aug.) 1927.

Treatment of Treponematous Ulcers with Bismuto-Yatren. R. P. Parsons, Am. J. Syph. **11**:425 (July) 1927.

Tschmarke's Treatment of Burns. K. Reschke, Arch. f. klin. Chir. **146**:763 (July 12) 1927.

Tuberculous Ulcerations of Skin Treated with Vaudremer's Vaccine. A. Sézary and E. Benoist, Bull. et mém. Soc. méd. d. hôp. de Paris **43**:1196 (July 28) 1927.

Ultraviolet Radiation in Dermatology. S. E. Dore, Brit. M. J. **2**:255 (Aug. 13) 1927.

Utilization of Skin of Upper Eyelid for Repair of Small Facial Defects. J. E. Sheehan, Arch. Otolaryng. **6**:107 (Aug.) 1927.

SYPHILOLOGY

Acetic Anhydride-Sulphuric Reaction for General Paresis. A. G. Duncan, J. Ment. Sc. **73**:419 (July) 1927.

Allergic Skin Reactions in Gonorrhea. E. Rajka, Dermat. Wchnschr. **85**:1138 (Aug. 13) 1927.

Antigens in Vernes Flocculation Test for Syphilis. M. A. Lyons and A. B. Baylis, Am. J. Syph. **11**:465 (July) 1927.

Aortic Signs of Infantile Syphilis. Martagão Gesteira, Arch. latino-am. de pediat. **21**:409 (June) 1927.

Bismuth Arsphenamine Sulphonate. John H. Stokes and Stanley O. Chambers, J. A. M. A. **89**:1500 (Oct. 29) 1927.

Bismuth Treatment of Syphilis. D. Lees, Brit. M. J. **2**:298 (Aug. 20) 1927.

Cardiovascular Syphilis. A. S. Warthin, Atlantic M. J. **30**:675 (Aug.) 1927.

Cerebral Lesions of Syphilis. A. Estupiñan, Bol. d. Hosp. civil de San Juan de Dios, **2**:30 (Feb.-April) 1927.

Changes in Spinal Fluid in Early Stage of Syphilis. B. Tashiro, Acta derm. **9**:559 (June) 1927.

Character of Cerebral Cerebrospinal Fluid in General Paralysis. H. Carlill and W. E. C. Dickson, Lancet **2**:275 (Aug. 6) 1927.

Combined Malaria Treatment of Syphilis. P. Mulzer, Deutsche Ztschr. f. Chir. **203**:654 (May) 1927.

Comparative Study of Colloidal Mastic and Colloidal Gold Tests on Cerebrospinal Fluids. R. C. Henderson, U. S. Veterans' Bureau M. Bull. **3**:803 (Aug.) 1927.

Comparison of Kahn, Meinicke, Kolmer-Wassermann and Ruediger-Wassermann Tests. E. H. Ruediger, Am. J. Syph. **11**:450 (July) 1927.
Comparison of 10,000 Wassermann and Kahn Tests Run in Parallel. L. D. Thompson and E. Ebel, J. Lab. & Clin. Med. **12**:1087 (Aug.) 1927.

Development Cycle of Treponema Pallidum. C. Levaditi et al., Bull. Acad. de méd. **98**:149 (July 26) 1927.
Device for Dilution of Antigen in Kahn Precipitation Test. H. Silvette, J. Lab. & Clin. Med. **12**:1096 (Aug.) 1927.
Diagnosis of Neurosyphilis. C. Mainini and J. Renner, Semana méd. **34**:469 (Aug. 25) 1927.
Dryer and Ward Sigma Reaction in Congenital Syphilis. H. E. Hall and F. M. B. Allen, Arch. Dis. Child. **2**:171 (June) 1927.

Experimental Syphilis: VII. A. M. Chesney, C. R. L. Halley and J. E. Kemp, J. Exper. Med. **46**:223 (Aug. 1) 1927.
Extent of Temporary Increase of Wassermann Reaction in Syphilis During Treatment. H. Haxthausen, Hospitalstid. **70**:616 (June 30) 1927.

Fever-Producing Methods in Treatment of General Paralysis. Editorial, J. A. M. A. **89**:1337 (Oct. 15) 1927.

Gastric Syphilis. K. Herman, Wien. klin. Wchnschr. **40**:1000 (Aug. 4) 1927.
General Paralysis: The Effect of Nonspecific Protein Therapy on the Blood and Spinal Fluid. M. M. Kunde, G. W. Hall and F. J. Gerty, J. A. M. A. **89**:1304 (Oct. 15) 1927.
Gold Sol Reaction in Syphilitic Cerebrospinal Fluid. E. Bender, Dermat. Wchnschr. **85**:1249 (Sept. 3) 1927.

Incidence of Syphilis at Shantung Christian University Dispensary. L. F. Heimburger, China M. J. **41**:541 (June) 1927.
Intraventricular Treatment of Paresis. Cestan, Riser and Pérès, J. Nerv. & Ment. Dis. **66**:227 (Sept.) 1927.

Kahn Reaction. C. E. Gabel, W. Virginia M. J. **23**:471 (Sept.) 1927.
Kahn's Reaction in Serodiagnosis of Syphilis. B. Abadjieff, Ztschr. f. Hyg. u. Infektionskrankh. **107**:742 (July 25) 1927.
Kahn Test in Experimental Syphilis. J. E. Walker, J. Infect. Dis. **41**:233 (Sept.) 1927.
Keloid Formation and Malignant Syphilis. E. Vollmer, Dermat. Wchnschr. **85**:1233 (Sept. 3) 1927.

Late Recurrence of Syphilis After Abortive Treatment. H. Hecht, Dermat. Wchnschr. **85**:1291 (Sept. 10) 1927.
Late Stages of Syphilis in Rabbit After "Symptomless Infection." R. Prigge, Deutsche med. Wchnschr. **53**:1340 (Aug. 5) 1927.
Localization of Spirochaeta Pallida in Brain in Progressive Paralysis. E. A. D. E. Carp, Nederl. Tijdschr. v. Geneesk. **71**:386 (July 23) 1927.

Malarial and Tryparsamide Treatment of Paresis. N. W. Kaiser and M. K. Amdur, Ohio. State M. J. **23**:656 (Aug.) 1927.
Malarial Treatment of General Paralysis. H. W. Bayly, M. J. & Record **126**:269 (Sept. 7) 1927.
Malaria Treatment of General Paralysis. C. W. Stone, Ann. Clin. Med. **5**:1119 (June) 1927.
Micro-Kahn Test in Syphilis. R. A. Kilduffe and W. W. Hersohn, J. Lab. & Clin. Med. **12**:946 (July) 1927.
Microprecipitation Test for Syphilis. M. G. Peterman, Am. J. Dis. Child. **34**:404 (Sept.) 1927.

Optimal Dose of Antigen for Wassermann Test. S. B. Hooker, J. Immunol. **14**:129 (Aug.) 1927.
Osteomyelitis of Long Bones in Infant with Inherited Syphilis. J. Thomson, Edinburgh M. J. **34**:466 (Aug.) 1927.

Pathologic Anatomy of Syphilis of Pulmonary Artery. S. M. Peck, Arch. Path. **4**:365 (Sept.) 1927.
Pathology and Therapy of Spirochetal Diseases. G. Steiner and J. Steinfeld, Klin. Wchnschr. **6**:1597 (Aug. 20) 1927.

Pitfalls of Wassermann Reaction. R. C. Jamieson and L. W. Shaffer, Am. J. Syph. **11**:456 (July) 1927.
Preventive Treatment of Syphilis. H. D. L. Spence, Lancet **2**:379 (Aug. 20) 1927.
Probable Syphilitic Origin of Certain "Albuminuric" Placentas. A. Fruhinsholz, Gynéc et Obstét. **15**:329 (May) 1927.
Pyrexia Induced by Intravenous Protein Therapy in General Paralysis. J. M. Mackenzie, Lancet **2**:223 (July 30) 1927.

Retinitis Pigmentosa Arrested by Organotherapy and Antisyphilitic Treatment. H. L. Hilgartner and J. S. Lankford, Endocrinology **11**:237 (May-June) 1927.

Simplification of Kolmer Test. J. B. Rucker, Jr., J. Lab. & Clin. Med. **12**:1109 (Aug.) 1927.
Spasmodic Associated Movements of Eyes: Can They be Produced by Syphilis? G. H. J. Pearson, Arch. Neurol. & Psychiat. **18**:414 (Sept.) 1927.
Spirochaeta Pallida in Lymph Glands in Earlier Stage of Frambesia. Y. Ikegami, Acta derm. **9**:558 (June) 1927.
Standardization of Wassermann Test. R. Gilbert and V. Langworthy, Am. J. Syph. **11**:475 (July) 1927.
Sulpharsphenamine Poisoning. C. D. Post, Am. J. Syph. **11**:444 (July) 1927.
Sulphur Treatment of Dementia Paralytica, etc. K. Schroeder, Ugesk. f. Laeger **89**:759 (Aug. 25) 1927.
Syphilis. R. E. Jameson, J. Iowa M. Soc. **17**:285 (Aug.) 1927.
Syphilis Among Negroes in South. J. E. Paullin, H. M. Davison and R. H. Wood, Boston M. & S. J. **197**:345 (Sept. 1) 1927.
Syphilis in Light of Recent Biologic and Immunotherapeutic Researches. S. Bergel, Med. Klin. **23**:1187 (Aug. 5) 1927.
Syphilis of Lung in Children. J. C. Navarro and E. A. Beretervide, Semana méd. **34**:471 (Aug. 25) 1927.
Syphilis of Stomach. C. Herman, Brit. J. Radiol. **32**:296 (Aug.) 1927.
Syphilis Transmitted by Transfusion. I. I. Levy and L. Ginsburg, Am. J. Syph. **11**:447 (July) 1927.
Syphilitic Chancre of Cervix and Pregnancy. Guyot et al., Bull. Soc. d'obst. et de gynéc. de Paris **16**:452 (July) 1927.
Syphilitic Nephritis. E. Hess, Am. J. Syph. **11**:438 (July) 1927.

Tertiary Congenital Syphilis. T. Hirsch, Zentralbl. f. Gynäk. **51**:2100 (Aug. 13) 1927.
Therapeutic Value of Hot Baths: Especially in Syphilis. J. F. Schamberg and H. W. Tseng, Am. J. Syph. **11**:337 (July) 1927.
Treatment of General Paralysis by Coley's Fluid. J. Rotter and J. A. Knoop, J. Kansas M. Soc. **27**:304 (Sept.) 1927.
Treatment of Neurosyphilis. J. L. McCartney, Am. J. Syph. **11**:398 (July) 1927.
Treatment of Syphilis. E. Flehme, Northwest Med. **26**:401 (Aug.) 1927.
Treatment with Tryparsamide and Mercury of 100 Cases of Neurosyphilis. G. W. Schelm, Illinois M. J. **52**:228 (Sept.) 1927.
Tryparsamide in Acute Syphilitic Meningitis. E. R. Smith, Am. J. Syph. **11**:432 (July) 1927.
Tryparsamide in General Paresis. G. H. Stevenson, Canad. M. A. J. **17**:787 (July) 1927.

Use of Antisyphilitic Remedies. S. L. Axford, J. Kansas M. Soc. **27**:258 (Aug.) 1927.
Use of Streptobacillary Vaccine in General Paralysis and Multiple Sclerosis. A. Sézary and A. Barbé, Progrès méd. **53**:1146 (July 23) 1927.

Vagary of Syphilis. J. R. McCord, Am. J. Syph. **11**:443 (July) 1927.
Vascular and Nervous Symptoms in Syphilis. J. Strandberg, Hygiea, Stockholm **89**:599 (Aug. 15) 1927.

Wassermann Antigen. L. G. Hadjopoulos and R. Burbank, J. Lab. & Clin. Med. **12**:973 (July) 1927.

INDEX TO VOLUME 16

The star (*) preceding the page number indicates an original article in THE ARCHIVES. No author entries are made except for original articles. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O and S, respectively.

PAGE

Acarus scabiei: its history *294
Achorion schönleinii, recovery from circulating blood 63
Acnitis (?); case for diagnosis 237
Acrodermatitis chronica atrophicans: See Atrophia
  perstans cured by autogenous vaccine ... 465
Acrodynia, paroxysmal, of infancy 764
Actinomycosis 76
  cervical 468
  roentgenotherapy in 625
  secondary, of abdominal wall 620
  treatment of, with iontophoresis 469
Adenoma sebaceum 657
  sebaceum acne 657
  sebaceum of Pringle type 617
Adenopathy, submental tuberculous; secondary ulcerative vegetating cutaneous tuberculosis 620
Advertising agent's questionnaires *460
Alexander, H. L.: Food allergy in Henoch's purpura *322
Allergy, itching of eyelids due to allergic reaction 757
  roentgen-ray, experiments on question of 626
Alopecia areata in tuberculous patient 618
  areata totalis 493
  congenital (complete) 510
  dermatitis exfoliativa with, complicating arthritis deformans 499
  pseudo-pelade (Brocq) 524
  syphilis and 226, 227
  syphilitic, tubercle of Mibelli and 210
  unilateral 491
  universalis 497
Aluminum, acetate, supposed dangers in wet dressings of *754
Aneurysm, arteriovenous 77
Angiolupoid of Bloch and Pautrier (?) 810
Angioma: See also Hemangiomatosis
  angioma 70
  cavernosum treated with radium 803
  multiple 244
  serpiginosum, comparison of progressive pigmentary dermatosis (Schamberg) with *141
  tumor of arm and, in new-born infant .. 622
  ulcerated, of face 468
Angiosarcoma apparently cured by electrocoagulation and roentgen rays 487
Antimony and potassium tartrate in chancroidal infections 462
Antipyrine, symmetrical eruption due to local use of, in patient with hyperhidrosis 620
Aortitis endocrine troubles with, in patient with congenital syphilis 202
  syphilitic, nature and distribution of lesions in 330
Argyria 68, 242
Arsenic: See also Arsphenamine
  findings in dermatologic conditions 614
  keratosis (arsenical) 485
  poisoning 801
  poisoning from oral administration of; sodium thiosulphate in 59
  psoriasis and 202
  therapy, effect of, in combination with bismuth, on Wassermann reaction 329
  therapy of eczema 614
Arsphenamine, dermatitis 86, 226, 238
  erythema induratum treated with neoarsphenamine 486
  erythroderma and polyneuritis due to ... 202
Arsphenamine—Continued PAGE
  hemorrhages of uterine mucosa and pituitary gland following injections of neoarsphenamine 623
  methods to reduce harmful effects of neoarsphenamine *442
  postarsphenamine hemorrhagic purpura .. 765
  stomatitis 336
Arteriosclerosis and livedo reticularis *431
Arteritis of dorsolumbar region, dry gangrene due to 616
Arthritis deformans, dermatitis exfoliativa with alopecia complicating 499
Atrophia, acrodermatitis chronica atrophicans 516, 782
  acrodermatitis chronica atrophicans and Hodgkin's disease 474
  acrodermatitis chronica atrophicans (Herxheimer-Pick), deep nerve lesion with tuberculoid perineural infiltration in case of 619
  acrodermatitis chronica atrophicans (Pick-Herxheimer) 617
  acrodermatitis chronica atrophicans (Pick-Herxheimer) in early stage 617
  cutis 371
  cutis idiopathica 353
  fibromatosis and, following zoster: diagnosis 349
  idiopathic 632
  syphilitic macular 61
  syphilitic optic, intracisternal injections in 757
Autodermotherapy, trial of, by electrocoagulation: 2 patients with psoriasis treated by 619

Bacillus of cutaneous gangrene 620
Balsam of Peru, dermatitis produced by .. *44
Barbital, erythema of ninth day due to 467
Bazin's Disease: See Erythema induratum
Bechet, P. E.: Lupus erythematosus, lupus vulgaris, tuberculids and tuberculosis of skin; treatment with gold compounds *563
  Scabies; small institutional epidemic with interesting and unusual features *51
Becker, S. W.: Melanin pigmentation: systematic study of pigment of human skin and upper mucous membranes, with special consideration of pigmented dendritic cells *259
Beeson, R. B.: Acarus scabiei: its history *294
Beinhauer, L. G.: Predisposing factors of eczema *12
Bismuth, fatality due to hemorrhagic nephritis in course of treatment with 467
  in lupus erythematosus 622
  intragluteal nodules of, edematous vesicular erythroderma cured by excision of . 622
  in treatment of congenital syphilis 759
  mercury and; new preparation of, in treatment of syphilis 205
  phosphate, neutral, in syphilis 768
  poisoning, fatal case of 628
  poisoning, in treatment of syphilis *456
  prolonged edematous vesicular erythroderma due to 467
  salt, insoluble, gangrenous tendency of severe local disturbances due to intermuscular injection of 620
  therapy, effect of, alone and in combination with arsenobenzenes, on Wassermann reaction 329
  therapy of psoriasis 627
  ulceration of tonsils 779
Blastomycosis ... 94, 94, 106, 211, 503, 788, 798
  effect of gentian violet on organism of blastomycotic infection *153
  erosive 470

INDEX TO VOLUME 16

PAGE

Blood, calcium content of, in skin diseases 200
chemistry, sulphur; thiemia in certain dermatoses and in secondary syphilis.. 467
hypercholesterinemia and lipomatosis.... 466
serum calcium in urticarias; ephedrine therapy *553
sugar, content of whole blood in syphilis *16

Book Reviews:

Atlas of Skin and Venereal Diseases; F. Schamberger and K. Hubschmann.. 373
Diagnostic des Principaux Cancers; H. Hartmann 373
Eminent Chemists of Our Time; B. Harrow 813
Handbuch der Haut-und Geschlechtskrankheiten; G. Arndt, B. Bloch and others 673
Practical Methods in the Diagnosis and Treatment of Venereal Diseases for Practitioners and Students; D. Lees.. 251
Syphilis Viscérales Tardives: M. Grenet, R. Levent and L. Pellissier..... 674
Bowen's Disease: See Dermatoses
Bromide intoxication 758
Bromine, in body fluids, simple color test for 462
Bromoderma 340, 487
treated with sodium chloride..... 809
vegetating; case 467
with lesions appearing on hard palate.. 221
Burgess, J. F.: Endogenous irritants as factors in eczema and in other dermatoses; preliminary report.....*131
Burns, from acid and alkali, treatment of. 337
Butyn, conjunctivitis and dermatitis due to 757, 758

Calcium cyanamide dermatosis..... 765
therapy, value of.....*555
Cancer: See also Epithelioma; Nevocarcinoma
after roentgen-ray treatment in vitro.... 625
Bowen's precancerous dermatosis...519, 661
Bowen's precancerous dermatosis followed by; case 204
breast, end-results in 563 cases..... 471
breasts, postoperative irradiation of..... 768
cutaneous, histologic diagnosis of.....*755
in Filipinos, incidence of..... 331
intra-oral, treatment with radium emanation 626
melanotic, in negro..... 667
methods and results in treatment of.... 626
of back of hand..... 230
of base of tongue..... 108
of bladder; scrotal elephantiasis; radiodermatitis 244
primary of right tonsil..... 225
propaganda 625
squamous cell, in old stasis ulcer..... 241
suprasyphilitic leukoplasia of uterus and tar, in rabbits..... 764
Cannon, A. B.: Lupus erythematosus; treatment with tuberculin..... 628
Castellani, A.: Fungi and fungous diseases *383, *571, *714
Cerebrospinal Fluid in differential diagnosis 58
Chancre, chancroidal infections, antimony and potassium tartrate in..... 462
meatal 75
multiple 74
of conjunctiva with parenchymatous keratitis 758
of lip 785
of nasal septum; 3 cases..... 462
of tongue 784
of tonsil 499
pseudo-syphilitic, due to cauterization with chronic acid..... 201
redux 235
secondary syphilid resembling..... 483
syphilitic, investigation of spirochetes in, during course of specific treatment... 764
tuberculous, in nursing infant..... 622
Chancroid, phagedenic, of abdominal wall, considered as lympogranulomatosis, of twenty-seven months' duration, cured in fifteen days by heterotherapy..... 204

PAGE

Charcot joint 484
Cheilitis glandularis 63, 222
Chickenpox, in monkeys; nuclear inclusions produced by varicella virus in testicles of monkeys..... 333
Chilblains, condition of legs somewhat resembling erythema induratum; 2 cases; case of erythema induratum for comparison 761
pernio 791, 794
Chondrodermatitis nodularis chronica helicus 62
Cleveland, D. E. H.: Raynaud's disease; 2 cases coexisting with other dermatoses (dermatitis herpetiformis, lupus erythematosus) *548
Collodion, flexible, use of tetraiodomethenamine in, in treatment of dermatophytosis *697
Colloid degeneration of skin.....*156, 250
Condyloma, condylomatous lupus of tongue 620
Conjunctivitis, dermatitis and, due to butyn 757, 758
Constipation, chronic, risk of liquid paraffin in 627
Copper sulphate, glycerin-alcohol solution of: note correcting report of May, 1926 (alcoholic copper sulphate in dermatology) 205
Copra itch, importance of symbiosis in.... 463
Cornbleet, T.: Inoculation tuberculosis of skin simulating sporotrichosis.....*312
Craver, L. F.: Extensive sarcoma of skin treated by unfiltered roentgen ray.... *35
Creeping disease 465
eruption, lesion of, produced by ant..... 79
Criticism, some criticism of us..... 196
Cummer, C. L.: Dermatitis produced by balsam of Peru..... *44
Cutis verticis gyrata; case..... 515
Cyst-adenomas, multiple 486
Cysts, mucous retention (?), case for diagnosis 480

Demodex impetigo (?); case for diagnosis. 237
Dermatitis: See also Acrodermatitis; Blastomycosis; Chrondrodermatitis; Neurodermatitis; Phenolphthalein dermatitis; Pyoderma; Radiodermatitis, etc.
allergic 514
chronic roentgen-ray 791
conjunctivitis and, due to butyn....757, 758
dry, of palms in hypothyroidism..... 245
due to feathers..... 773
due to pictorial papers..... 777
exfoliativa 228
exfoliativa in negro..... 505
exfoliativa, mild, after ingestion of 0.5 Gm. of treparsol, 5 years after postarsphenamine erythroderma 618
exfoliativa, with alopecia, complicating arthritis deformans..... 499
facial, of bacterial origin, and glossitis with leukoplastic patches..... 622
hat band 811
herpetiformis 486, 497, 784, 796
herpetiformis (?); case for diagnosis.... 247
herpetiformis, coexisting with lupus erythematosus and Raynaud's disease.....*548
herpetiformis, (herpes gestationis)..... 355
intense primrose, of bullous type..... 764
medicamentosa: See under names of drugs, as Antipyrine; Arsenic; Arsphenamine; Balsam of Peru; Gold; Phenolphthalein
medicamentosa (tentative diagnosis).... 500
nodular 97
of Jacquet 233
on hands 788
primrose 769
psychologic factor in..... 60
radiodermatitis; scrotal elephantiasis; carcinoma of bladder..... 244
radiodermatitis, unusual case of, and neuritis 201
repens 95
roentgen-ray, cases of..... 513
seborrheic 237, 792

PAGE

Dermatitis—Continued
subacute (eczema) (?) 250
venenata 346
venenata, chronic 106
venenata (inecto) 528
venenata in rubber tire industry 766
Dermatologists, some criticism of us 196
Dermatology, index to current literature 122, 252, 377, 534, 678, 817
modern, some requirements of, in this country; development of specialty ... *539
Dermatolysis 248
(molluscum type) 809
Dermatophytosis: See Mycosis
Dermatoses, Bowen's precancerous ... 519, 661
Bowen's precancerous, followed by cancer; case 204
from insufficient menstruation 628
intolerance to sugar as factor in production of 333
professional 765
progressive pigmentary (Schamberg's Disease) 246, 490
progressive pigmentary (Schamberg); review of literature; two cases; comparison with angioma serpiginosum and purpura annularis telangiectodes. *141
sympathetic radiotherapy in; results in 150 cases 206
thiemia in, and in secondary syphilis ... 467
tropical, in West Indies *53
Diabetes, study of skin in 500 cases 760
Diathermo-Coagulation, high-frequency apparatus for 202
Dyskeratosis 615

Ebert, M. H.: Livedo reticularis *426
Ecthyma following varicella 249
Eczema 342, 638, 771
acute infectious eczematoid dermatitis .. 105
arsenical, treatment 614
eczematiform streptococcides 615
endogenous irritants as factors in; preliminary report *131
infantile, relief of itching by epinephrine 463
infectious eczematoid dermatitis and tertiary syphilis 225
in tomato peelers 627
predisposing factors of *12
relation of excretion of sugar in sweat to *706
roentgen-ray treatment of 625
(sensitization), neurodermitis 639
Edema, solid, macrochilia 509
Electrocoagulation, epithelioma treated by, histology of 465
trial of autodermotherapy by; 2 patients with psoriasis treated by 619
Elephantiasis, in West Indies *53
scrotal; radiodermatitis; carcinoma of bladder 244
vulvar 622
Endocrines, congenital syphilis and; rôle of biologic tests 252
troubles of, with aortitis in patient with congenital syphilis 202
Endothelioma in infant 762
Ephedrine therapy; blood serum calcium in urticarias *553
Epidermolysis bullosa ... 104, 109, 218, 636, 637
bullosa acquisita 776
bullosa, lichen planus and 666
Epidermophytosis, need for research in treatment of 759
Epinephrine, relief of itching by, in infantile eczema 463
Epithelioma 249, 341
adenoides cysticum (Brooke); case 334
basal cell 518
basal cell, of morphea type 790
canalicular, with Paget's disease of breast 466
diagnosis value of lattice fibers in *25
granuloma inguinale simulating 778
histology of, treated by electrocoagulation 465
lupus erythematosus and 345

PAGE

Epithelioma—Continued
morphea-like 790
multiple benign 780
of mouth 796
of penis: 5 cases 759
of shaft of penis 478
sclerodermatous 655
squamous cell, of hand 484
squamous cell, of tongue 485
superficial, of skin, treated by mixed method 624
Erosio interdigitalis 503
Erysipelas, dosage of quartz lamp treatment of 628
experimental, local immunity in 332
treatment of acute infectious and contagious diseases, with especial reference to scarlet fever, erysipelas, measles and meningitis 58
Erythema, ab igne 363, 363, 781
centrifugal annular 623
chronic, of legs 62
due to ultraviolet rays (artificial sunburn); plaques of leukoderma following 205
elevatum diutinum 107, 250
elevatum diutinum (Crocker); case 514
figuratum perstans (?); case for diagnosis 236
induratum 71, 86, 214, 235
induratum, Bazin's Disease, syphilis (?) 635
induratum, chilblainy condition of legs somewhat resembling; 2 cases; case of erythema induratum for comparison ... 761
induratum; embolic tuberculosis 241
induratum, of giant and dwarf types, both accompanied by papulonecrotic tuberculids; 2 cases 203
induratum, papulonecrotic tuberculid and 785
induratum treated with neoarsphenamine 486
induratum tuberculosis colliquativa (?) 364
induratum, tuberculous cervical adenitis and lichen scrofulosorum 786
induratum with positive Wassermann reaction 511
multiforme 240, 246
multiforme, erythema nodosum (unilateral) with lesions like 786
multiforme bullosum 77, 213
multiforme (iris type) 505
multiforme, recurrent (transition approaching dermatitis herpetiformis) accompanied by urticarial outbreaks .. 466
multiforme, toxic eruption of pregnancy 509
nodosum, its place in medicine 760
nodosum (unilateral) with erythema multiforme-like lesions 786
of ninth day, due to barbital 467
papular, urticarial type of toxic origin .. 641
skin, roentgen-ray dose based on "R" unit 626
toxic 110, 504
Erythrocyanosis, symmetrical submalleolar 619
Erythroderma, edematous vesicular, cured by excision of intragluteal nodules of bismuth 622
following single injection of iodobismuthate of quinine 623
polyneuritis and, due to arsphenamine .. 202
post-arsphenamine, mild exfoliative dermatitis after 618
prolonged edematous vesicular, due to bismuth 467

Erythromelia, of Pick, with lichen planus of tongue and buccal mucosa 619
Erythrosis, pigmentary facial (peribuccal pigmentary erythrosis of Brocq) 620
Eyermann, C. H.: Food allergy in Henoch's purpura *322
Favus, of scalp, with indeterminate lesion on body 354
Feathers, dermatitis due to 773
Fibromatosis and atrophy following zoster 349
Fibrosarcoma 504

PAGE

Folliculitis decalvans 66, 98
trichophytic, treatment of kerion and certain cases of, by intravenous injections of Lugol's solution..... 615
Foot and Mouth Disease, physical and chemical properties of virus of; description of strain used; titration and centrifugation experiments 333
physical and chemical properties of virus of; resistance to chemicals..... 333
Foreign Body tumor 797
Fox, H.: Tropical skin disease in West Indies *53
Fox-Fordyce disease 784
Frazier, C. N.: Effect of elevation of body temperature on course of experimental syphilis in rabbit..... *445
Fungi and fungous diseases...*383, *571, *714
infection; scroll-like lesions on chest.... 792
"yeastlike," simple scheme of classification for use of clinicians and clinical pathologists *579

Gangrene, cutaneous 620
dry, due to arteritis of dorsolumbar region 616
penile ulcerations: diagnosis and pathogenesis 620
tendency to, of severe local disturbances due to intermuscular injection of insoluble bismuth salt..... 620
Gans, O.: Skin picture in hematopoietic diseases *1
Glossitis with leukoplastic patches, and facial dermatitis of bacterial origin.. 622
Gold: See also under Thiosulphates
compounds, treatment with, in lupus erythematosus, lupus vulgaris, tuberculids and tuberculosis of skin.....*563
dermatitis medicamentosa and lupus erythematosus 794
lupus erythematosus discoideus treated with 795
lupus erythematosus treated with..... 798
sodium thiosulphate and, lupus erythematosus treated with.....358, 358
Goodman, H.: Suggested nomenclature for ultraviolet zones *291
Granuloma annulare 672
annulare: cure following vaccine therapy 205
case for diagnosis..... 239
coccidiodes *308
fungoides: See also Mycosis fungoides
fungoides with leukemic blood findings.. 244
inguinale 59, 67, 109
inguinale simulating epithelioma..... 778
inguinale tropicum 502
mycotic 73
of unknown type..... 810
pyogenicum; case 201
Granulosis rubra nasi..... 618
Grebin, Z. N.: Comparative value of methods to reduce harmful effects of neoarsphenamine by different vehicles *442
Greenbaum, S. S.: Blood serum calcium in urticarias; with note on ephedrine therapy *553
Gumma, multiple of legs..... 227
of sternoclavicular articulation..... 488
of tonsil 77, 208
on dorsum of hand..... 488
syphilitic 507
ulcerating of sole..... 240
Guy, W. H.: Granuloma coccidioides.....*308
Hair, epilation by thallium acetate..... 766
Halloran, C. R.: Mycetoma in American negro; case *611
Hemangiomatosis 81
Hematopoietic system diseases, skin picture in *1
Hematoporphyrinuria, skin eruption from exposure to sunlight..... 470
Hemorrhages of uterine mucosa and pituitary gland following injections of neoarsphenamine 623
under skin 792

PAGE

Herpes, discussion on "The Pathological Relationships of the Herpetic Diseases" 60
facialis, recurrent 242
gestationis: See Dermatitis, herpetiformis
herpetic diseases 59
of cheek, recurrent, results of radiotherapy 617
pathologic relationships of herpetic diseases from clinical standpoint..... 60
virus, preservation of..... 766
zoster 664
zoster, fibromatosis and atrophy following 349
zoster, gangrenous, of glans penis..... 616
zoster, left thoracic, simultaneous appearance of, and paralysis of left seventh, and then of third, fourth and fifth segments, in tuberculous patient with tabes; latent syphilis aroused by zoster 204
zoster, varicella and, by biotropism..... 616
zoster, vasodilatation arising from antidromic impulses, to herpes zoster and trophic effects 472
Herxheimer reaction, local, in case of vitiligo 623
Highman, W. J.: Use of tetraiodomethenamine in flexible collodion in treatment of dermatophytosis.....*697
Hodgkin's Disease: See Lymphogranulomatosis
Hypercholesterolemia, xanthomatosis cutis and 762
Hyperchromia of face of type of chloasma in course of debilitating secondary syphilis 202
Hyperhidrosis, keratosis palmaris et plantaris 643
symmetrical eruption due to local use of antipyrine in patient with..... 620
Hyperkeratosis, follicular; Riehl's melanosis, oil papules and, in metal lathe worker 206

Ichthyosis 525, 653
Impetigo circinata (?); case for diagnosis 222
contagiosa, treatment of, by ultraviolet rays 767
herpetiformis 466
Infection, skin, new experiment of..... 198
Inguinal glands, action of rays on..... 767
Intestines, disturbances of, endogenous irritants as factors in eczema and in other dermatoses; preliminary report..*131
Iodoform, influence of, on Wassermann reaction in rabbits..... 767
Ionization, iodine, ultraviolet rays and, sclerodermatous plaque much improved by combined treatment with..... 619
Iontophoresis, treatment of actinomycosis with 469
Itch, straw (?)..... 645
Jacob, F. M.: Granuloma coccidioides....*308
Jaundice, acholuric (familial or hereditary), splenomegaly with 242
Kahn Test, comparison with Wassermann test in 15,000 examinations..... 59
for syphilis in comparison with Wassermann results 614
report of 300,000 462
significance of 331
Keloids 343
Keratitis, parenchymatous, chancre of conjunctiva with 758
Keratoderma, blennorrhagic: See Keratosis, blennorrhagic
palmare hereditarium 233
palmaris and plantaris..... 799
plantaris and palmaris..... 799
Keratoma palmare et plantare hereditarium 487
Keratosis: See also Dyskeratosis; Parakeratosis; Tubercle of Mibelli
arsenical 485
blennorrhagic, keratoderma blennorrhagica 219, *741
blennorrhagic, keratoderma blennorrhagicum, with changes in nails..... 366

PAGE

Keratosis—Continued
fixed pigmentary deposit and, following quartz light therapy; case..... 762
follicularis 615
Mibelli's tubercle 494
palmaris et plantaris.....247, 642, 642
palmaris et plantaris, hyperhidrosis..... 643
Kerion, trichophytic folliculitis and, treated by intravenous injections of Lugol's solution 615
Kesten, B. M.: Treatment of urticaria with ephedrine *189
Krasnow, F.: Sugar content of whole blood in syphilis *16

Labadie, J. H.: Colloid degeneration of skin; report of case..... *156
Lacayo, J. R.: American origin of syphilis; with citations from early Spanish authors collected by Dr. Montejo y Robledo *683
Leishmania tropica, treatment of tropical sore due to 463
Lentigo, generalized (?)..... 84
Leprosy.....224, 334, 360, 367, 473, 810
acute manifestations of.....337, 338
contagion and transmission in..... 768
in West Indies..... *53
macular 104
mixed: cutaneous and nervous; negative Wassermann reactions..... 616
morphea atrophica: its possibly leprous origin 469
negative: nasal lesions..... 471
negative: persistence of bacilli in deep foci 471
negative: relapses 471
nodular 810
ocular 758
temporary anisocoria in patient..... 757
tuberculin in treatment of..... 338
Leukemia (chronic, lymphatic)..... 365
congenital, hereditary 86
congenital, hereditary; correction..... 195
cutis (?)..... 475
fragility of white blood cells..... 329
leukemid 95
Leukoderma: See also Vitiligo
appearance of plaques of, following erythema due to ultraviolet rays (artificial sunburn) 205
Leukoplakia, associated melanotic patches of buccal mucosa and 622
buccalis 772
lingual, rhabdomyoma of tongue in syphilitic patient with..... 617
of tongue and buccal mucosa..... 75
patches of, and facial dermatitis of bacterial origin, glossitis with 622
suprasyphilitic of uterus and cancer.... 764
Leukosis, skin picture in hematopoietic diseases *1
Lichen corneus hypertrophicus, accompanying, radioresistant; lichen planus cured by medullary radiotherapy 617
nitidus 623, 761
scrofulosorum 85, 250
scrofulosorum, tuberculous cervical adenitis and erythema induratum..... 786
trichophyticus: kerion celsi 477
urticatus (?); case for diagnosis..... 245
Lichen, Planus....347, 669, 782, 789, 789, 812
annular 243, 249
annularis in patient with tertiary syphilis 477
atrophicus 342
atrophicus linearis 651
atrophicus, probable, of zosteriform distribution in child aged 3 years..... 622
buccal 621
case for diagnosis..... 239
cured by medullary radiotherapy; accompanying radioresistant lichen corneus hypertrophicus 617
cured by treparsol..... 621
epidermolysis bullosa and..... 666
erythematosus, generalized, poikiloderma or 789

PAGE

Lichen, Planus—Continued
hypertrophicus 368
hypertrophicus (?); case for diagnosis.. 248
incipient 499
juveniles 232
late results of treatment of, by medullary radiotherapy 618
of mucous membranes..... 336
of penis 235
of tongue and buccal mucosa, erythromelia of Pick with 619
of unusual type..... 224
psoriasis and 783
superficial radiotherapy and..... 621
with hyperpigmentation 348
with lesions of mucous membrane..... 508
with or without radiotherapy; buccal lichen planus with cutaneous eruption (cutaneous reaction), with or without radiotherapy 621
Light: See also Sunlight; Ultraviolet Rays desensitization to, and pyrocatechin intravenously 621
influence of, on growth and malignancy of transplanted neoplasm of rabbit.... 332
influence of, on reaction to infection in experimental syphilis 332
Lingua geographica (?); case for diagnosis. 245
Lipomatosis and hypercholesterinemia..... 466
Lipskeroff, I. B.: Comparative value of methods to reduce harmful effects of neoarsphenamine by different vehicles. *442
Livedo reticularis *426, 663
reticularis and tuberculid..... 101
Lugol's solution, treatment of kerion and certain cases of trichophytic folliculitis by intravenous injections of..... 615
Lupoids, miliary, and tuberculids of face. 618
miliary, disseminated 466
Lupus, condylomatous, of tongue..... 620
pernio, with adenopathy and osseous lesions 469
vulgaris 646, 670, 785
vulgaris, extensive 225
vulgaris healed with injections of gold thiosulphate 473
vulgaris, hypertrophic 664
vulgaris, late syphiloderm simulating.... 243
vulgaris, lupus erythematosus, tuberculids and tuberculosis of skin; treatment with gold compounds.....*563
vulgaris of upper eyelid and brow..... 623
vulgaris; pityriasis rubra pilaris..... 246
vulgaris treated with pyrogallic acid.... 487
vulgaris, variations in surface tension of urine in 629
Lupus Erythematosus.....64, 76, 233
243, 246, 343, 362, 489, 506, 512, 523, 529, 660, 772, 787, 787
bismuth in 622
coexisting with dermatitis herpetiformis and Raynaud's disease.....*548
congestive type 233
dermatitis medicamentosa (gold) and... 794
discoides 93
discoides (recurrent) 93
discoideus treated with gold..... 795
disseminatus 87
disseminatus (subacute type)..... 88
epithelioma and 345
extensive, treated with sodium thiosulphate 804
healed 659
lupus vulgaris, tuberculids and tuberculosis of skin; treatment with gold compounds *563
mycosis fungoides and 800
poikiloderma (?) 217
psoriasis simulating 245
treated with gold 345, 798
treated with gold and sodium thiosulphate 358, 358, 806
treatment with tuberculin..... *8
Wassermann reaction in, carried out with large amounts of serum..... 621
Lymphadenosis, skin picture in hematopoietic diseases *1

PAGE

Lymphangioma 230, 507, 783
circumscriptum 665
of hand 81
tuberosum of scalp..... 204
Lymphangitis, case for diagnosis..... 522
Lymphogranulomatosis, acrodermatitis chronica atrophicans and Hodgkin's disease. 474
Hodgkin's disease 780
Hodgkin's disease of skin and mucous membranes 330
Hodgkin's disease with skin manifestations 521
malignant, of abdomen and pelvis (Hodgkin's disease of abdominal type)..... 466
occurrence of leukopenia in Hodgkin's disease 330
phagedenic chancroid of abdominal wall, considered as; of twenty-seven months' duration, cured in fifteen days by heterotherapy 204

MacKee, G. M.: Some requirements of modern dermatology in this country; development of specialty.....*539
Maduromycosis: See Mycetoma
Malaria, therapeutic: See Neurosyphilis; Paralysis, General
general paresis and 619
Meinicke Reaction, flocculation reaction in pregnant women 206
Melanocarcinoma in xeroderma pigmentosum 764
Melanoderma (?) 781
parasitic; Vagabonds' disease and generalized melanoderma 617
Melanosis in railroad grade worker who handled ties treated with creosote.... 206
Melanotic carcinoma in negro..... 667
patches of, associated with leukoplakia of buccal mucosa 622
Riehl's, oil papules and follicular hyperkeratosis in metal lathe worker..... 206
Menstruation, insufficient, skin diseases as sequelae of 338, 628
Mercury, bismuth and; new preparation of, in treatment of syphilis..... 205
vapor lamp, treatment of purpura hemorrhagica by 758
Mikulicz's Disease, mycosis fungoides and. 812
Mora, J. M.: Bismuth poisoning in treatment of syphilis.....*456
Morphea: See Scleroderma, circumscribed
Morrissey, M. J.: Morvan's disease.....*166
Morvan's Disease: See Syringomyelia
Mumford, P. B.: Xeroderma and abnormalities of skin texture.....*317
Mycetoma in American negro; case.....*611
maduromycosis (due to Aspergillus nidulans) 91
Mycosis: See also Actinomycosis; Blastomycosis; Epidermophytosis; Mycetoma; Oidiomycosis; Otomycosis; Sporotrichosis; Trichophytosis, etc.
dermatophytosis 207
fungoides.....118, 521, 526, 781, 798
fungoides in negro..... 213
fungoides, Mikulicz's disease and..... 812
interdigital lesions of mycotic origin..... 616
use of tetraiodomethenamine in flexible collodion in treatment of dermatophytosis *697
Myelitis, acute syphilitic.....764
Myelosis, skin picture in hematopoietic diseases *1

Nails, leukonychia 359
oidiomycosis of 62
onychomycosis 802
onychomycosis successfully treated by roentgen ray 808
onychoptosis, recurrent, occurring as family disorder 762
Neoarsphenamine: See Arsphenamine
Neoplasm, cells of, radiosensitivity of..... 624
Nephritis, hemorrhagic, fatality due to, in course of treatment with bismuth..... 467

PAGE

Netherton, E. W.: Tularemia with reference to its cutaneous manifestations; report of case *170
Neuritis, radiodermatitis and; unusual case. 201
Neurodermatitis, axillary (Fox-Fordyce)... 616
case for diagnosis..... 496
eczema (sensitization) 639
Neurofibroma 523
Neurofibromatosis, Recklinghausen's Disease 371
Neurosyphilid 655
Neurosyphilis, pregnancy and 614
secondary syphilid with..... 784
therapy, by malaria..... 462
therapy, malaria, as preventive of..... 616
Neurotropism of acquired syphilis in patients with congenital syphilis..... 203
Nevocarcinoma, treatment of, by electrocoagulation 204
Nevus: See also Angioma
anemicus 369
angiomatosus, involving entire right hand and forearm 351
case for diagnosis..... 527
cerebelliform (?)..... 118
flammeus 81
linear (?) case for diagnosis..... 246
nevus 82
or xanthoma (?); case for diagnosis..... 244
unius lateralis (verrucosus)..... 82
vascularis 76
verrucose 235, 811, 811
verrucosus (linearis) 82
white fibrous cicatricial, with angiomatous border 616
woolly, hereditary, of scalp..... 337
zosteriform, with comedones 468
Newspapers, dermatitis due to pictorial papers 777
Nitritoid crises precipitated by applications of cryotherapy (solid carbon dioxide); urticaria pigmentosa 203

Obituary: Hartzell, Milton Bixler, 1854-1927 54
Odessaer Dermatologische und Venerologische Gesellschaft, twenty-fifth anniversary of 55
Oil papules, Riehl's melanosis and follicular hyperkeratosis in metal lathe worker. 206
Ornstein, G. G.: Lupus erythematosus; treatment with tuberculin..... *8
Oroya Fever, etiology of; experiments on cross-immunity between verruga peruana and 333
etiology of; pathologic changes observed in animals experimentally infected with bartonella bacilliformis; distribution of parasites in tissues; response of skin of macacus rhesus and anthropoid apes to inoculation with bartonella bacilliformis 332
Osteitis, radiation 625
Osteoperiostitis, syphilitic, of left tibia.... 76
Otomycosis, 3 cases..... 463
relative frequency 58

Paget's Disease, of breast, beginning, with underlying canalicular epithelioma.... 466
Paraffin, liquid, risk of, in chronic constipation 627
Paraffinoma 783
Parakeratosis variegata (?) 506
Paralysis, of left seventh, and then of third, fourth and fifth segments, appearing simultaneously with left thoracic zoster, in tuberculous patient with tabes; latent syphilis aroused by zoster..... 204
general, clinico-pathologic study; significance of so-called typical laboratory findings 330
malaria and 619
malaria treatment of; histopathologic observations in 15 cases..... 58
treated by injections of brand of acetarsone (Stovarsol); 2 cases..... 201
Paraplegia, syphilitic, occurring 7 months after chancre 468

PAGE

Parapsoriasis 484, 647, 648, 671
erythrodermie pityriasique en plaques disseminées 647
in syphilitic patient; case..... 203
lichenoides 647
lichenoid (?); case for diagnosis....216, 245
lichenoides in patient with syphilis..... 76
Parathyroid and skin..... 765
Pellagra (?) 799
Pemphigus 668, 776, 788
beginning in mouth, with retarded cutaneous manifestations 201
case for diagnosis.....236, 645
foliaceus 344, 780, 802
foliaceus; apparent recovery..... 654
of both eyes..... 757
vulgaris....111, 517, 780, 781, 783, 792, 800
Periadenitis mucosa necrotica..... 231
Phenolphthalein dermatitis in colored patient 498
dermatitis medicamentosa 353, 486, 488, 781, 795
eruption 356, 370
eruption (?), case for diagnosis..... 479
Phototherapy for radium and roentgen-ray injuries of skin 625
Pigmentation: See also Melanosis
melanin; systematic study of pigment of human skin and mucous membranes, with special consideration of pigmented dendritic cells 259
of buccal mucosa 468
of face and neck..... 790
unusual type 84
Pityriasis rosea, experimental transmission of *185
rosea, microsporid 361
rosea, of scalp: almost invariable localization in children..... 200
rosea, with initial plaques on face, following wasp-bite 204
rubra pilaris 234, 344
rubra pilaris (Devergie) (?)..... 527
rubra pilaris; lupus vulgaris..... 246
versicolor, treatment of by rubs..... 623
Plaques, transitory benign, of tongue..... 662
Pneumococcus III, production of purpura by derivatives of: nature of purpura producing principle 333
Poikiloderma (?) 117
generalized lichen planus erythematosus, or 789
lupus erythematosus (?) 217
Polyneuritis, erythroderma and, due to arsphenamine 202
Poradenolymphitis, case of..... 615
Potassium tartrate and antimony in chancroidal infections 462
Pregnancy, flocculation reaction in pregnant women 206
neurosyphilis and 614
serologic statistics of maternity hospital of Toulouse for 1926..... 205
toxic eruption of, multiform erythema type 509
Prurigo, chronic, of face..... 616
circinate 619
nodularis 239, 650
Pruritus of anus and vulva, of long standing, cured by correction of uterine retroversion; familial congenital syphilis. 203
vulvar, treatment of..... 765
Pseudoxanthoma elasticum: See under Xanthoma
Psoriasis 237, 342, 770
actinodermatitis (radium) 529
arsenic and 202
autodermotherapy by electrocoagulation in, trial of 619
bismuth treatment of 627
extensive, occurring in man aged 65.... 782
guttate 507
hyperkeratotic, with papillomatosis and cutaneous horns 622
in negro 108
lichen planus and..... 783
psoriasiform syphilis 363
seborrheic type 212
simulating lupus erythematosus..... 245

PAGE

Psoriasis—Continued
unusual type 107
vulgaris, variations in surface tension of urine in 629
Purpura, annularis telangiectodes. 350, 354, 798
annularis telangiectodes, comparison of progressive pigmentary dermatosis (Schamberg) with *141
hemorrhagica (essential thrombocytopenia): treatment by mercury vapor lamp 758
Henoch's, food allergy in.....*322
postarsphenamine hemorrhagic 765
production of, by derivatives of pneumococcus III: nature of purpura-producing principle 333
simplex, chronic, of lower extremities... 623
Pyoderma (?); case for diagnosis..... 247
Pyrocatechin intravenously and desensitization to light 621
Pyrogallic acid, lupus vulgaris treated with 487

Quartz lamp treatment of erysipelas, dosage 628
light therapy, keratoses and fixed pigmentary deposit following; case..... 762
Quinine, iodobismuthate of, severe erythroderma following single injection of.... 623
Quinlan, R. V.: Teeth in cases of congenital syphilis; roentgen-ray studies of unerupted teeth *605

Rabinowitch, I. M.: Excretion of sugar in sweat; its relationship to eczema....*706
Radioactive ointment, treatment of chronic ulcers with 616
Radiodermatitis 525
Radiosensitivity of neoplastic cells..... 624
Radiotherapy: See also Phototherapy; Quartz lamp; Sunlight; Ultraviolet Rays
angioma cavernosum treated with radium. 803
angiosarcoma apparently cured by electrocoagulation and 487
biologic unit of radiation..... 625
dose of roentgen ray to produce skin erythema based on "R" unit..... 626
idiosyncrasies of radiation; irregular and delayed reactions 627
in cancer; methods and results..... 626
in intra-oral cancer, with radium emanation 626
in recurrent herpes of cheek..... 617
injuries to skin by radium and roentgen-ray; phototherapy for 625
irradiation, biologic action of..... 624
latent period following, before biologic reaction 626
lichen planus cured by medullary radiotherapy, and lichen corneus hypertrophicus resistant to radiotherapy..... 617
lichen planus with or without; buccal lichen planus with cutaneous eruption, with or without..... 621
medullary, late results of treatment of lichen planus by..... 618
of cancer in vitro..... 625
of eczema 625
of superficial epitheliomas of skin..... 624
of sympathetic system in skin diseases.. 472
onychomycosis successfully treated by... 808
roentgen-ray allergy 626
roentgen-ray dermatitis 513
superficial, lichen planus and..... 621
sympathetic, in dermatoses: results in 150 cases 206
with small doses 624
Radium therapy: See Radiotherapy
Rats, hairless 668
Raynaud's Disease, coexisting with dermatitis herpetiformis and lupus erythematosus; 2 cases.....*548
with ocular complications..... 757
Recklinghausen's Disease: See Neurofibromatosis
Reynolds, H. S.: Morvan's disease.....*166
Rhabdomyoma of tongue in syphilitic patient with lingual leukoplakia..... 617

PAGE

Rhinoscleroma 791
Rice, J. P.: American origin of syphilis; with citations from early Spanish authors collected by Dr. Montejo y Robledo *683
Ringworm: See also Favus
hairs on scalp, detection of, by their fluorescence under ultraviolet light.... 762
infection of all nails of hands..... 802
kerion celsi: lichen trichophyticus..... 477
of scalp treated by thallium acetate epilation 463
tinea capitis in adult..... 477
treatment of, by thallium acetate..... 766
Roentgen Rays, chronic dermatitis due to.. 791
energy radiated as, from Coolidge tube.. 624
method of measuring, particularly in skin therapy 624
studies of unerupted teeth in congenital syphilis *605
therapy: See Radiotherapy
Roentgenology in North America..... 625
Rosacea with accompanying conditions..... 765
Rosen, I.: Sugar content of whole blood in syphilis *16
Roseola, recurrent 350, 784
Rostenberg, A.: Keratoderma blennorrhagicum; critical review with report of case *741
Rubber, dermatitis venenata in rubber tire industry 766
hypersensitiveness to 768

Sanderson, E. S.: Effect of genitan violet on organism of blastomycotic infection. *153
Sarcoid.....91, 214, 238, 241, 343
case for diagnosis..... 652
multiple benign (Boeck)..... 492
of eyelids 757
oil tumors or, cause of growth of..... 331
Sarcoma: See also Angiosarcoma; Fibrosarcoma
case for diagnosis..... 220
extensive, of skin, treated by unfiltered roentgen ray *35
Kaposi's 94, 109
melanotic 64
Scabies, acarus scabiei: its history..... *294
antiscabetic treatment of pityriasis versicolor 623
small institutional epidemic with interesting and unusual features..... *51
Scarlet Fever, blanching phenomenon..... 768
intradermal salt solution test in..... 330
Scars, deforming, following unsuccessful plastic operation 811
Schamberg's Disease: See Dermatosis, progressive pigmentary
Sclerodactyly, progressive scleroderma and, with endocrine troubles..... 202
Scleroderma 69, 357, 664
case for diagnosis 478
circumscribed: basal cell epithelioma of morphea type 790
circumscribed, morphea 528
circumscribed, morphea atrophica: its possibly leprous origin 469
circumscribed, morphea in colored man.. 493
circumscribed: morphea-like epithelioma. 790
circumscribed, morphea with involvement of mucosa of lip..... 240
circumscribed; white spot disease..... 801
of scalp in band.....618, 618
plaque of, much improved by combined treatment with ultraviolet rays and iodine ionization 619
progressive, and sclerodactyly, with endocrine troubles 202
sclerodermatous epithelioma 655
symmetrical, tending to extend over upper extremities 201
syphilis and 334
Scrofuloderma 78
Seborrhea: See Dermatitis, seborrheic
Septicemia, tertiary syphilitic..... 204
Sharlit, H.: Use of tetraiodomethenamine in flexible collodion in treatment of dermatophytosis *697

PAGE

Silver, H.: Keratoderma blennorrhagicum; critical review with report of case...*741
Skin, colloid degeneration of; case.....*156
degeneration of 644
heredity as applied to..... 470
idiosyncrasy of 469
infection, new experiment of..... 198
texture, abnormalities of, and xeroderma of *317
vascular reactions of, to injury..... 471
Smith, D. C.: Effect of gentian violet on organism of blastomycotic infection...*153
Societies, Canadian, directory of.....
.....119, 256, 374, 531, 675, 814
local, directory of.....
.....119, 256, 374, 531, 675, 814
national, directory of.....
.....119, 256, 374, 531, 675, 814
SOCIETY TRANSACTIONS:
American Dermatological Association.... 630
Brooklyn Dermatological Society.....
.....515, 770, 798
Chicago Dermatological Society..81, 360, 508
Detroit Dermatological Society.....239, 504
Manhattan Dermatological Society.....
.....207, 345, 494, 792
Minnesota Dermatological Society.....
.....228, 785
New England Dermatological Society.64, 339
New York Academy of Medicine, Section on Dermatology and Syphilis.....
.....70, 216, 473, 774
New York Dermatological Society.....
.....111, 350, 519, 801
Philadelphia Dermatological Society....
.....78, 235, 489, 666
Pittsburgh Dermatological Society...105, 528
San Francisco Dermatological Society... 513
St. Louis Dermatological Society.....
.....210, 499, 662
Sodium chloride, bromoderma treated with 809
Spinal Fluid, ill effects from lumbar puncture, with report of post-puncture fatality 331
Spirocheta, investigation of, in syphilitic chancre during course of specific treatment 764
pallida, in spinal fluid of syphilitic patients 337
Splenomegaly, acholuric jaundice (familial or hereditary jaundice) with..... 242
Sporotrichosis 80, 110
inoculation tuberculosis of skin simulating *312
Staphylococcus infection 341
Stomatitis 93
arsphenamine 336
chronic erosive, of infectious origin (?); case for diagnosis..... 236
cryptococco-bacillaris, importance of symbiosis in 463
thrush of conjunctiva..... 758
Streptococcus in eczematiform eruptions.. 615
infections of epidermis..... 465
Sugar, intolerance to, as factor in production of some dermatoses..... 333
Sunlight, action on lymphoid organs..... 767
Sweat Glands: See also Hyperhidrosis; Syringoma
excretion of sugar in its relationship to eczema *706
sudorimeter, simple form of..... 761
Symbiosis, importance of, in causation of diseases and symptoms of disease..... 463
Syphilid, aggravated by irradiation (?); case for diagnosis..... 243
annular (?); case for diagnosis..... 249
annular, small 505
developing during treatment..... 371
late syphiloderm simulating lupus vulgaris 243
nodular 507
palmar 797
papular, containing spirochetes, negative serologic reactions in patient with... 467
papulosquamous 250
(psoriasiform type) 99
rupial 784

PAGE

Syphilid—Continued
secondary cutaneous, and tabes, coexistence of 615
secondary, resembling chancre..... 483
secondary, with neurosyphilis..... 784
Syphilis 366
acquired, neurotropism of, in patients with congenital syphilis..... 203
acute syphilitic myelitis..... 764
alopecia and 226, 227
American origin of, with citations from early Spanish authors collected by Dr. Montejo y Robledo.....*683
Bazin's Disease 635
beginning in lymph nodes; secondary papular chancre; resistance to intensive treatment 203
bismuth poisoning in treatment of.....*456
congenital 797
congenital, bismuth treatment of..... 759
congenital, endocrine glands and: rôle of biologic tests..... 202
congenital, endocrine troubles with aortitis in patient with..... 202
congenital, teeth in; roentgen-ray studies of unerupted teeth.....*605
congenital, 202 cases..... 760
congenital, unrecognized cases of..... 61
corymbose type 530
experimental, effect of elevation of body temperature on course of.....*445
experimental, fever treatment of..... 628
experimental, influence of light on reaction to infection in..... 332
familial congenital; pruritus of anus and vulva, of long standing, cured by correction of uterine retroversion..... 203
Hutchinson's teeth *606
in children, insufficiently treated..... 769
intracisternal injections in syphilitic optic atrophy 757
late 67, 214
late secondary papular lesions resembling chancre 227
latent, aroused by left thoracic zoster appearing simultaneously with paralysis of left seventh, and then of third, fourth and fifth segments, in tuberculous patient with tabes..... 204
leukemoid blood picture in..... 337
livedo reticularis and.....*428
mottled chin of..... 484
neutral bismuth phosphate in..... 768
new preparation of bismuth and mercury in treatment of 205
of central nervous system, treatment.... 759
of larynx, active, coexisting with active pulmonary tuberculosis 614
or tuberculosis 515
paraplegia occurring 7 months after chancre 468
pleuropulmonary, 3 masked forms of: paucilobar pneumopathy, predominantly bronchial; insular mutilating corticopleuritis; slate-colored syphilitic pneumonia (sclerogummatous corticopleuritis) 465
primary, experimental, in rabbits; therapeutic effect of hot baths in..... 58
psoriasiform 363
regression of, and arsphenamine..... 769
reinfection 227, 774
rupial 215
scleroderma and 334
secondary 64
secondary, debilitating; hyperchromia of face of type of chloasma in course of. 202
secondary, thiemia in certain dermatoses and 467
secondary, with negative serology..... 468
secondary, with tertiary manifestations.. 75
simple method of testing for, based on reducing power of serums..... 621
spirochaeta pallida in spinal fluid in.... 337
statistics of cases of, seen in houses of tolerance in Parisian district from 1917 to 1926 205

PAGE

Syphilis—Continued
sugar content of whole blood in..... *16
syphilitic alopecia and tubercle of Mibelli 210
syphilitic "spina-ventosa," with radiography; case 204
tertiary, infectious eczematoid dermatitis and 225
tertiary, lichen planus annularis in patient with 477
tertiary, of eyelid..... 757
tertiary syphilitic septicemia..... 204
tests for, in Illinois.....*461
undergoing clinical evolution with silent serology 466
(unusual) 481
Syphiloderm, nodular circinate, of face and scalp 785
Syphilology, index to current literature.... .....127, 254, 380, 536, 680, 820
Syringocystoma 206
Syringoma 350, 667
Syringomyelia (Morvan's disease).....72, *166

Tabes, and secondary cutaneous syphilids, coexistence of 615
Tar carcinomas in rabbits..... 628
painting with, skin and organ changes in white mice after..... 629
Teeth, Hutchinson's *606
in congenital syphilis; roentgen-ray studies of unerupted teeth.....*605
Telangiectasia, generalized 241
of unknown origin..... 492
Temperature, body, elevation of, in experimental syphilis *445
Templeton, H. J.: Progressive pigmentary dermatosis (Schamberg); with review of literature, report of 2 cases and comparison with angioma serpiginosum and purpura annularis telangiectodes.*141
Tetraiodomethenamine use of, in flexible collodion in treatment of dermatophytosis *697
Thallium 464
acetate epilation, ringworm of scalp treated by 463, 766, 766
Thiosulphate, gold, lupus erythematosus treated with 806
gold, lupus vulgaris healed with injections of 473
sodium, gold and, in treatment of lupus erythematosus 358, 358
sodium, lupus erythematosus treated with 804, 806
Thrombo-angiitis obliterans in women..... 766
Thrush: See under Stomatitis
Thymus, action of rays on..... 767
Thyroid Gland in dermatology...764, 764, 765
Tinea: See Pityriasis versicolor; Ringworm
Tongue, black, hairy (lingua nigra pilosa). 368
burning 506
hairy 486
lesions on 791
Treparsol, lichen planus cured by..... 621
Trichloracetic acid, treatment of rodent ulcer by 471
Trichomycosis nigra, importance of symbiosis in 463
rubra, importance of symbiosis in..... 463
Trichophytosis, chronic, of skin and nails in all members of family, with invasion of mucosae, lymph nodes and bone.763, 763
of dorsum of foot..... 467
pubic and crural, due to "trichophyton persicolor" 206
Trichotillomania 340, 669
Tropical Sore: See Leishmania tropica
Trypanosomes, experiments on production of Wassermann reagins by means of.. 332
Tubercle bacilli, in tuberculids..... 469
of Mibelli, syphilitic alopecia and..... 210
Tuberculid 634
classification and treatment of..... 472
leg ulcer in girl of 13, treated without success as tuberculid; negative serology; rapid cure with arsphenamines.. 202
livedo reticularis and..... 101

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PAGE

Tuberculid—Continued
lupus erythematosus, lupus vulgaris and tuberculosis of skin; treatment with gold compounds *563
of face, and miliary lupoids..... 618
papulonecrotic 232, 491
papulonecrotic, accompanying erythema induratum of giant and dwarf types; 2 cases 203
papulonecrotic, erythema induratum and 785
papulonecrotic, of upper and lower extremities and ulcerative tuberculids of legs 618
ulcerative, of legs, and papulonecrotic tuberculids of upper and lower extremities 618
Tuberculin, in treatment of leprosy..... 338
Tuberculosis, buccal 621
colliquativa 364, 783
condylomatous lupus of tongue due to.. 620
cutis 248
embolic; erythema induratum..... 241
erythema induratum tuberculosis colliquativa (?) 364
inoculation, of skin simulating sporotrichosis *312
livido reticularis and..... *429
nonsyphilitic; paradoxic inactivation of Wassermann reaction in patients with 205
of skin, lupus erythematosus, lupus vulgaris, and tuberculids; treatment with gold compounds *563
of tongue 83, 513
pulmonary, active, coexisting with active syphilis of larynx..... 614
scrofuloderma gummosa (tuberculosis colliquativa) 512
secondary ulcerative vegetating cutaneous; submental tuberculous adenopathy 620
syphilis or 515
tuberculous cervical adenitis, erythema induratum and lichen scrofulosorum.. 786
tuberculous chancre in nursing infant.. 622
ulcerative, of lips..... 83
verrucosa cutis 232, 807
verrucosa cutis in negro..... 646
Tularemia, with reference to its cutaneous manifestations; case *170
Tumors: See also Angioma; Cancer; Epithelioma; Fibrosarcoma; Granuloma; Keloids; Lymphangioma; Mycetoma; Myelosis; Neurofibroma; Paraffinoma; Rhabdomyoma; Sarcoid; Syringocystoma; Syringoma, etc.
epithelial, in white mice after painting with tar 629
foreign body 797
lymphoid, of neck..... 653
of face 620
oil, or sarcoids, cause of growth of..... 331
subungual 627
Ulcer, acute, of vulva..... 617
bismuth, of tonsils..... 779
chronic, treatment of, with radioactive ointment 616
chronic, ultraviolet ray treatment..... 767
factitious 657
of forearm 100
gangrenous penile: diagnosis and pathogenesis 620
lesion on forehead..... 788
of leg, in girl of 13, treated without success as tuberculid; negative serology; rapid cure with arsphenamines..... 202
perforating, of heel..... 782
rodent, treatment by trichloracetic acid. 471
syphilitic gummatous, grouped in inguinal region 618
tuberculous, of leg..... 490
varicose, of leg..... 628
venereal, vaccine therapy of..... 63
Ulerythema ophryogenes 494
Ultraviolet Rays, action on lymphoid organs 767
iodine ionization and, sclerodermatous plaque much improved by combined treatment with 619
treatment in chronic ulcers..... 767

PAGE

Ultraviolet Rays—Continued
treatment of impetigo contagiosa by.... 767
xeroderma pigmentosum treated with.... 639
zones, suggested nomenclature for.....*291
Urine, variations in surface tension of, in skin diseases 629
Urticaria, blood serum calcium in; ephedrine therapy *553
chronic 212
due to cold..... 619
papular 61
pigmentosa 113, 653
pigmentosa, adult type..... 468
pigmentosa, bullous type..... 468
pigmentosa; nitritoid crises precipitated by applications of cryotherapy (solid carbon dioxide) 203
treatment of, with ephedrine.....*189
variations in surface tension of urine in 629
wheal production through internal channels 329
Usher, B.: Excretion of sugar in sweat; its relationship to eczema.....*706

Vaccination, statement on, by surgeon generals 199
Vagabond's Disease: See Melanoderma, parasitic
Varicella and zoster by biotropism..... 616
Vasodilatation arising from antidromic impulses, to herpes zoster and trophic effects 472
Verruca, herbs for..... 201
peruana, experiments on cross-immunity between oroya fever and..... 333
plana juvenilis 339
Vitiligo 666
local Herxheimer reaction in case of... 623

Wassermann Reaction, comparison with Kahn test in 15,000 examinations..... 59
effect of bismuth alone, and in combination with arsenobenzenes, on..... 329
experiments on production of Wassermann reagins by means oy trypanosomes 332
influence of iodoform on, in rabbits..... 767
in lupus erythematosus, carried out with large amounts of serum..... 621
paradoxic inactivation of, of nonsyphilitic tuberculous patients 205
results, Kahn precipitation test for syphilis in comparison with..... 614
serologic statistics of maternity hospital of Toulouse for 1926..... 205
Way, S. C.: Lattice fibers; diagnostic value in epithelioma..... *25
Whitehouse, H. H.: Lupus erythematosus, lupus vulgaris, tuberculids and tuberculosis of skin; treatment with gold compounds *563
Wien, M. S.: Inoculation tuberculosis of skin simulating sporotrichosis.....*312
Wile, U. J.: Experimental transmission of pityriasis rosea; preliminary report...*185
Williams, H. U.: American origin of syphilis, with citations from early Spanish authors collected by Dr. Montejo y Robledo *683

Xanthoma 77
diabeticorum: unusual process of involution 329
nevus or (?); case for diagnosis..... 244
pseudoxanthoma elasticum; case..... 201
tuberosum multiplex 250
xanthelasma 96, 486
Xanthomatosis, cutis 370
cutis and hypercholesterolemia..... 762
Xeroderma 779
abnormalities of skin texture and.....*317
pigmentosum 666
pigmentosum, late 203
pigmentosum, melanocarcinoma in..... 764
pigmentosum, treated with ultraviolet light radiation 639

Yaws, importance of symbiosis in..... 463
in West Indies.....*53

CONTENTS

Page

The American Origin of Syphilis, with Citations from Early Spanish Authors Collected by Dr. Agnello Rossello. Herbert U. Williams, M.D., Buffalo, with the Collaboration of John P. Rice, Ph.D., Buffalo, and Joseph Renato Lacaye, M.D., Grenada, Nicaragua 681

The Use of Tetraiodomethenamine in Flexible Collodion in the Treatment of Dermatomycosis. Herman Sharlit, M.D., and Walter James Highman, M.D., New York 697

Excretion of Sugar in Sweat: Its Relationship to Glycuria. B. Usher, M.D., and I. M. Rabinowitch, M.D., Montreal 706

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Keratoderma Blennorrhagicum: Critical Review with a Report of a Case. Adolph Rosenberg, M.D., and Henry Silver, M.D., New York 771

Editorials:

Supposed Dangers in Wet Dressings of Aluminum Acetate 734

The Histologic Diagnosis of Cutaneous Cancer 735

Abstracts from Current Literature 737

Society Transactions:

Brooklyn Dermatological Society 770

The New York Academy of Medicine, Section on Dermatology and Syphilis 774

Minnesota Dermatological Society 785

Manhattan Dermatological Society 792

Brooklyn Dermatological Society 798

New York Dermatological Society 801

Book Reviews 813

Directory of Dermatologic Societies 814

Index to Current Literature 817

General Index 821